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A

MANUAL OF OTOLOGY

FOR

STUDENTS AND PRACTITIONERS

BY

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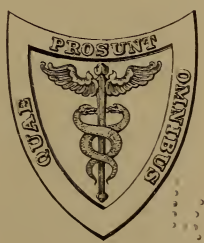

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PREFACE.

Many years' experience in teaching otology to undergraduates and to postgraduate students who are adopting it as a specialty has made the author conversant with its inherent difficulties. In the preparation of this manual it has been his aim to lighten these difficulties and to supply data which will enable those who thoroughly master them to become capable aurists. The simpler and less serious affections, which fortunately form the larger part of aural practice, have been discussed in full detail as to their diagnosis and treatment, and the orderly sequence in which these topics are taken up should afford the reader a properly correlated conception of the entire subject.

In describing operative procedures applicable to the middle ear and mastoid, the consideration of many minutiæ is essential. Careful adherence to the technic advised, with due attention to the measures indicated to avoid dangers and accidents, will insure the surgeon's becoming a safe and efficient operator. The chapter on Suppurative Diseases of the Labyrinth sets forth the present knowledge of the graver affections of this complex region. It is

hoped that this part of the subject has been presented in so clear and definite a manner as to bring it even within the easy grasp of the beginner.

The author desires to express his thanks to Dr. E. B. Dench, with whom he has been many years associated in aural work. He is also under obligations to Mr. K. K. Bosse for his work on the illustrations, and to the publishers, Messrs. Lea & Febiger, for their care in making the book.

C. E. P.

New York, 1916.

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MANUAL OF OTOLOGY.

CHAPTER I.

ANATOMY AND PHYSIOLOGY OF THE EAR.

The ear may be divided into three parts: the external, middle, and internal. The external and middle are concerned in transmitting sonorous vibrations and are therefore called the sound-conducting mechanism. The inner ear consists of the acoustic and static labyrinths. The acoustic labyrinth (the cochlea) with its central connections receives and analyzes sonorous vibrations, and is called the sound-perceiving mechanism; while the static labyrinth (consisting mainly of the semicircular canal system) with its central connections, is concerned in the maintenance of equilibrium.

EXTERNAL EAR.

The external ear is that part of the auditory apparatus which is situated external to the membrana tympani, and consists of the auricle and auditory canal or meatus.

Auricle.—The auricle or pinna is an irregular, oval-shaped appendage, composed of a cartilaginous framework covered with integument. It is attached to the side of the head at an angle so that its posterior surface is directed internally, and its anterior surface is directed externally. Its posterior surface is comparatively smooth, while its anterior surface is irregular and more or less concave, thus acting to collect sound waves and conduct

them into the external auditory canal. It presents numerous eminences and depressions. These vary in different subjects, both in size and position, but it is usually not difficult to identify those shown in the cut (Fig. 1), a study of which conveys a very good impression of the name and location of these eminences and depressions.

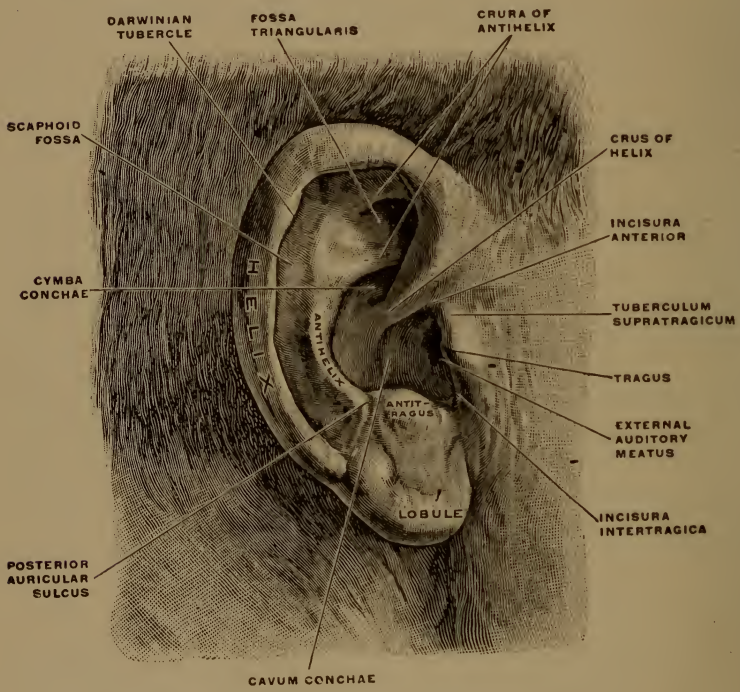

FIG. 1.—Anterior and external view of the auricle. (Gray.)

The muscles of the ear are rudimentary in man and not of surgical importance. The auricle is attached to the temporal bone by three ligaments—the superior, anterior and posterior—which extend from the cartilaginous framework of this structure to the bone forming the entrance to the external auditory canal.

The blood supply of the auricle is received through the

posterior auricular from the external carotid, the anterior auricular and some smaller branches from the temporal.

The nerves are the auricularis magnus from the cervical plexus, the auriculotemporal, which is a branch of the inferior maxillary nerve and some small branches of the small occipital nerve.

Meatus.—The external auditory canal is about one and a half inches long if measured from the tragus. A little more than the outer half is cartilaginous and membranous, the remaining inner part being osseous. If measured from the bottom of the concha, it is about one inch in length, of which the outer third is cartilaginous. The cartilage extends deeper on the anterior than on the posterior wall and is traversed by two vertical fissures (the fissures of Santorini) through which infective processes or purulent accumulations may extend from the canal to the tissues anterior to the ear. The membranous and osseous portions of the meatus form an obtuse angle with each other, the outer part having a direction upward, backward, and inward; while that of the inner part is downward, forward, and inward. The integument lining the outer part contains the openings of the ceruminous glands, and from it project the hairs of the meatus. These structures are found only in the cartilaginous, never in the osseous portion of the canal—a point of practical importance in the study of certain diseases affecting the external auditory canal. Slightly internal to the junction of the two parts the canal is usually contracted by the prominence of the anterior bony wall. This constricted portion is called the isthmus. Internal to this the canal usually widens anteriorly, forming a depression which extends to the membrana tympani. This renders it difficult at times to obtain a view of the extreme anterior part of the drum membrane, and also affords a lodgement for foreign bodies from which their extraction may become difficult. The membrana tympani forms the fundus of the canal and lies obliquely, thus making the canal deeper anteriorly and inferiorly than above and

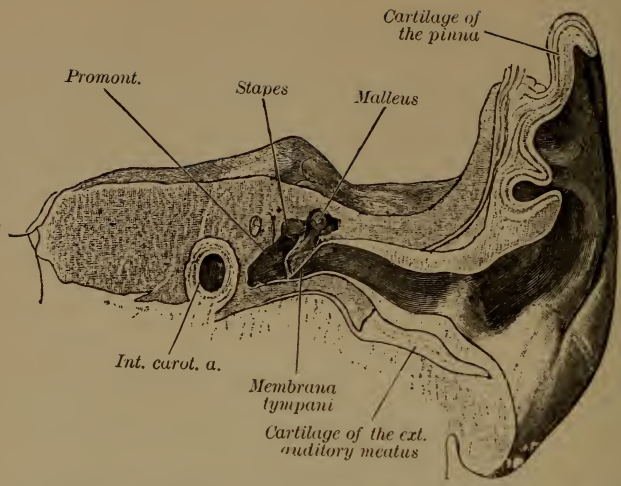

Fig. 2.—Horizontal section of the external auditory meatus and tympanum. (Gray.)

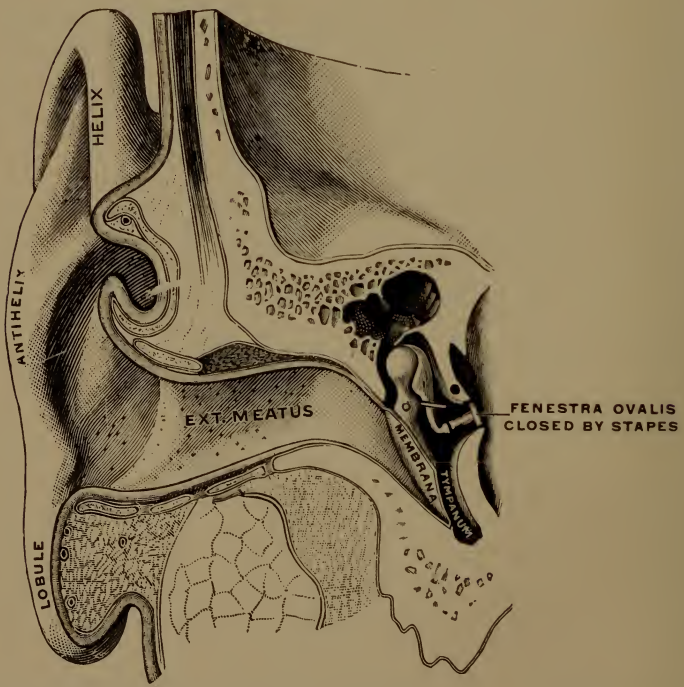

Fig. 3.—Vertical section of the external auditory meatus and tympanum. (Gerrish.)

posteriorly (Figs. 2 and 3). The osseous canal is formed principally by the outgrowth of the tympanic ring. At birth this ring forms an incomplete circle (Fig. 4). The part between the ends of the ring is called the segment of Rivinus, and is filled in by the squamous portion of the temporal bone. In the development of the bone this grows *pari passu* with the outgrowth from the tympanic ring and completes the canal above. This takes place

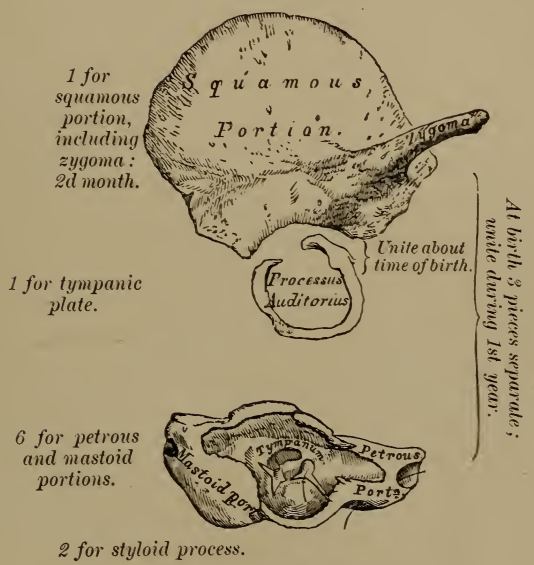

FIG. 4.—Tympanic ring. (Gray.)

in extra-uterine life. At birth there is no osseous canal, the tympanic ring, and therefore the drum membrane attached to it, lie practically on the surface. The inclination of the drum head is as great or slightly greater than in adult life, its external surface being directed downward, forward, and outward. The membranous canal is attached directly to the tympanic ring and its direction is upward and outward, the auricle being placed higher on the squama than in the adult.

The nerve supply of the external auditory canal is derived from the auriculotemporal, the auricularis magnus, and the pneumogastric through its auricular branch (Arnold's nerve). It is claimed that the facial supplies sensory and trophic filaments to the external auditory canal and auricle. This claim is based largely upon pathological investigations which are discussed under Herpes Zoster Oticus.

Tympanic Membrane.—The tympanic membrane forms the fundus of the external canal and part of the external wall of the tympanum and is therefore common to the external and middle ear. It is divided into two parts which are very unlike in structure and appearance. These are the membrana tensa and the membrana flaccida or Shrapnell's membrane. The membrana tensa is that part which is attached to the tympanic ring; while Shrapnell's membrane fills in the Rivinian segment, having its peripheral attachment at the internal end of the squamous portion of the canal. The membrana tensa consists of three layers—an outer one of epithelium which is continuous with that lining the meatus; a middle one of fibrous tissue, the fibers of which are arranged in either a radiating or circular manner and enclose the manubrium; while the inner layer is composed of mucous membrane which is continuous with that lining the tympanic cavity. Shrapnell's membrane is much smaller, and is really an extension of the skin of the superior canal wall, lined to a greater or less extent on its inner surface with the tympanic mucous membrane.

MIDDLE EAR.

Tympanum.—The middle ear lies internal to the tympanic membrane. It is an irregularly cuboidal cavity and presents six walls for examination: namely, internal, external, anterior, posterior, inferior or floor, and superior or roof. The cavity lies obliquely so that the internal wall is also partly superior. This must be remembered,

but for purposes of description is ignored as leading to confusion. The internal wall (Fig. 5) is principally formed of the bony labyrinth and presents five points for examination, as follows: (1) Superiorly, the prominence of the anterior end of the horizontal semicircular canal. (2) Immediately below this and extending horizontally is a ridge which is the external wall of the tympanic part of the Fallopian canal containing the

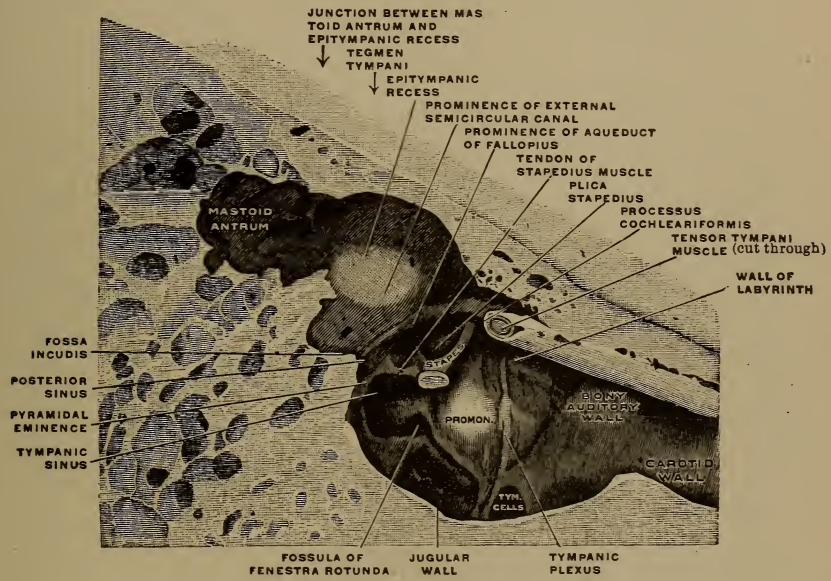

FIG. 5.—Internal tympanic wall. (Gray.)

facial nerve. (3) Immediately below this is the oval window at the bottom of a depression or niche. In the recent state the niche is occupied by the stapes, the foot-plate of which, together with its annular ligament, closes in the oval window. The shell of bone between the facial nerve and the oval window is very thin and sometimes absent—a point of surgical importance. (4) Below and behind the oval window, separated from it

by a process of bone which is part of the promontory, is the niche of the round window at the bottom of which is the fenestra rotunda, or round window. This is closed in by a thin membrane called the membrana tympani secondaria. (5) The promontory which lies between and anterior to the two fenestræ is of ivory-like bone, and a part of the labyrinthine capsule which forms the first cochlear whorl or turn. Anterior to this promontory the internal wall is receding, and the transverse diameter of the tympanum greater. The second cochlear whorl in this location does not form a prominence as the first.

The outer wall is formed by the tympanic membrane, the tympanic ring, and above by the internal end of the superior wall of the external auditory meatus. The anterior wall is formed internally by the bone covering the carotid canal, while externally is the opening of the Eustachian tube, and above this and separated from it by a bony process, the processus cochleariformis, is the canal for the tensor tympani muscle. The posterior wall of the tympanum is absent above on account of the opening into the mastoid antrum (additus ad antrum), on the floor of which is the fossa incudis in which lies the short process of the incus. Below is the pyramid, a hollow bony process, which contains the stapedius muscle. The tendon of this muscle emerges at the apex of the pyramid and is attached to the posterior crus of the stapes. At times there is a recess leading back into the bone from the posterior wall, called the sinus tympani.

The roof, or tegmen tympani, separates the tympanic cavity from the middle cranial fossa. It is a thin shell of bone and part of the inner table. A few small fragile cells may be found in this situation or the bone may present defects or dehiscences, thus allowing the middle ear to come into still closer relation with the cranial cavity. The tegmen is continuous posteriorly with the roof of the antrum, and anteriorly with the bone covering in the canal for the tensor tympani muscle. The floor or inferior wall of the tympanum is in relation with the

jugular bulb. The thickness of the bone in this situation varies. It may be very thin or even absent. If absent, the dome of the jugular bulb may extend into the tympanic cavity. When this condition is present there is danger of wounding the vessel in performing the operation of myringotomy. This has occurred, but fortunately this anomaly, which renders this accident possible, is rare. The floor is perforated by a small opening near the internal tympanic wall for the passage of the tympanic branch of the glossopharyngeal (Jacobson's nerve).

Eustachian Tube.—The Eustachian tube connects the tympanum with the nasopharynx. It is about one and a half inches in length. Its external one-half inch is osseous and lies adjacent to the tympanic cavity. The remaining part of the tube, about one inch in length, is membranous and cartilaginous, and is adjacent to the nasopharynx. At the junction of these two parts the lumen of the tube is contracted and the isthmus is thus formed. The inner end is trumpet-shaped, the pharyngeal orifice being much larger than the lumen at the isthmus. Its direction is downward, forward and inward, the amount of obliquity varying more or less with the shape of the skull. The tube is lined with mucous membrane, covered with ciliated epithelium, continuous with the mucous membrane of the tympanum and nasopharynx. Immediately above the tube and separated from it by a thin shell of bone—the processus cochleariformis—is the tensor tympani muscle which arises from the base of the temporal bone and adjacent part of the Eustachian tube, and is inserted into the manubrium.

Ossicles.—The tympanic cavity contains the ossicular chain (Fig. 6). This consists of the malleus, incus, and stapes. The malleus or hammer (Fig. 7) presents a head, neck, long and short process and handle for examination. The handle or manubrium lies in the tympanic membrane and partakes of the movements of this membrane and affords attachment to the tensor tympani muscle. The short process is a small conical process

Fig. 6.—Chain of ossicles and their ligaments, seen from the front in a vertical, transverse section of the tympanum. (Gerrish.)

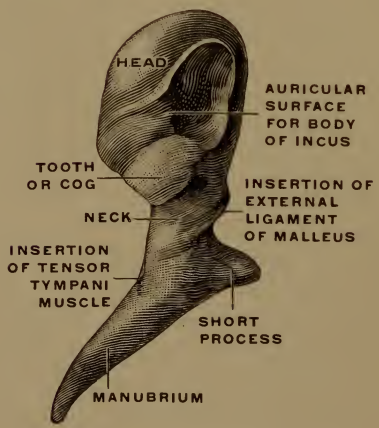

Fig. 7.—Posterior view of the right malleus. (Gray.)

surmounting the upper end of the handle and is directed outward. The long process lies in the Glaserian fissure. The neck is the constricted portion of bone connecting the handle with the head of the ossicle. The latter articulates with the incus. This articulation is of a rachet nature and allows the handle to make excursions outward without acting upon the incus, but movement inward causes the joint to bind and the incus to be moved. This is brought about by the teeth or cogs, which separate when the manubrium makes an extensive movement outward, but come in contact upon inward movement of the handle of the malleus. The incus (Fig. 8) presents

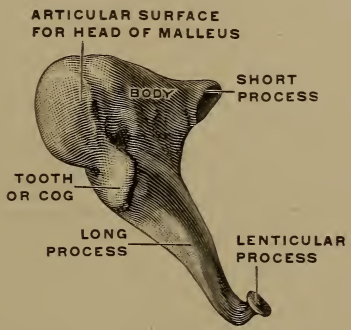

FIG. 8.—Mesial and front view of the right incus. (Gray.)

a body which articulates with the head of the malleus, a short process which lies in the fossa incudis, and a long process which is connected with the capitellum of the stapes, forming the incudostapedial articulation. The stapes (Fig. 9) or stirrup has a head or capitellum articulating with the long process of the incus, two crura, an anterior and posterior, which connect the head with the foot-plate. To the posterior crus is attached the tendon of the stapedius muscle. The foot-plate with its annular ligament fills in the oval window, forming what is termed the stapediovestibular articulation.

Ossicular Ligaments.—The ossicles are held in position by various ligaments which connect them with the

tympanic walls and with each other. The ligaments which connect the malleus with the tympanic wall are the anterior, external, and superior. The anterior consists of two parts, the band of Meckel and the anterior ligament of Helmholtz. The band of Meckel was formerly considered as a muscle and described as the laxator tympani. It is of fibrous tissue and is attached at the base of the long process and extends through the Glaserian fissure to the spine of the sphenoid. The anterior ligament of Helmholtz extends from the neck of the malleus to the anterior margin of the Rivinian segment. The external ligament extends from the junction of the head and neck of the malleus to the posterior part of the Rivinian segment. The posterior fibers

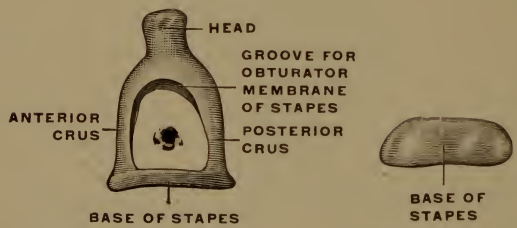

FIG. 9.—The right stapes, viewed from above. (Gray.)

of this ligament constitute the posterior ligament of Helmholtz and together with the anterior ligament of Helmholtz form what is termed the axis band. The superior or suspensory ligament is a small ligament extending between the head of the malleus and the tegmen tympani. The short process of the incus is held in position in the fossa incudis by a band of fibrous tissue. The ossicles are connected with each other at their articulations by fibrous tissue arranged more or less in the form of annular ligaments.

Mechanism of Ossicular Movement.—The axis band above described constitutes the axis upon which the movement of the ossicular chain takes place. The mechanism is a lever the long arm of which is the manu-

brium and the short arm the long process of the incus, the ratio of the arms being as three to two. The effect of this mechanism is therefore to diminish the amplitude but to increase the intensity of sonorous vibrations in transmitting them from the membrana tympani and consequently the manubrium to the stapes, and through it to the labyrinthine fluid. The tensor tympani, by its contraction, depresses and renders the drum membrane tense, causing it to assume a proper condition to be affected by sonorous vibrations. The action of the stapedius muscle is antagonistic to the tensor tympani, as its contraction acts to draw the foot-plate of the stapes away from the labyrinthine fluid, thereby diminishing intralabyrinthine pressure. Acting thus it protects the delicate structures within the labyrinth against loud noises or force exerted inward upon the drum membrane.

Mucous Membrane.—The mucous membrane of the middle ear is continuous with that in the tube and through it with the nasopharyngeal mucous membrane. It lines the tympanic cavity, the internal surface of the drum membrane, and is continuous with the membrane in the mastoid antrum and cells. It covers the ossicles and their ligaments and in so doing several vascular folds are formed. The anterior fold of the malleus extends from the anterior end of the Rivinian segment to the upper end of the handle of the malleus, and envelops the chorda tympani nerve, the long process of the malleus and the anterior ligament. The posterior fold extends from the upper end of the manubrium to the posterior margin of the Rivinian segment, and envelops the posterior tympanic part of the chorda tympani, and the external ligament of the malleus. Both of these folds have free concave margins and the depressions lying between the folds and the tympanic membrane are called the pouches or recesses of von Tröltsch, anterior and posterior respectively. Prussak's space lies between Shrapnell's membrane externally, the neck of the malleus internally, and the external ligament posteriorly. Besides

those described above there are various other folds, some more or less constant, others peculiar to the individual subject, which are of importance from their role in pathological middle-ear processes. Throughout the greater part of the tympanic cavity the mucous membrane is covered with columnar ciliated epithelium. That covering the internal surface of the drum membrane and the ossicles is of the squamous non-ciliated variety.

Arteries.—The arterial supply of the tympanum is quite free and derived from various sources. The tympanic branch of the internal maxillary artery enters through the Glaserian fissure and anastamoses with the stylomastoid branch of the posterior auricular which gains access to the tympanum through the Fallopian canal. Besides these there are the petrosal branch of the middle meningeal which enters through the hiatus Fallopii, some small branches of the middle meningeal entering through the petrosquamosal fissure, branches of the carotid reaching the tympanic cavity through the anterior tympanic wall, and the tympanic branch of the ascending pharyngeal which enters the tympanum through the foramen for Jacobson's nerve.

Nerves.—The nerve supply is derived from the tympanic branch of the glossopharyngeal which receives branches from the carotid plexus of the sympathetic. These are the caroticotympanic and small deep petrosal nerves which enter through the anterior tympanic wall. The tympanic plexus also receives branches from the superficial petrosal nerves. The plexus thus formed lies in grooves upon the internal tympanic wall and sends branches to the various structures in the middle ear and, according to some authors, a branch to the chorda tympani (*Ramus communicans*). The stapedius is supplied by a branch from the facial. The tensor tympani receives its nerve supply from the otic ganglion.

Facial Nerve.—The facial nerve as it passes through the temporal bone is of great interest to the otologist,

both from the symptoms arising from its involvement and the danger of its being injured in surgical procedures. The deep origin of the facial is from a nucleus in the floor of the fourth ventricle, although the fibers supplying the occipitofrontalis, the orbicularis palpebrarum and the corrugator supercilii, have a different origin and join the nerve before it leaves the brain. They are, therefore, not involved in nuclear facial paralysis. In the internal auditory meatus the nerve lies above and anteriorly, being separated from the eighth nerve by the pars intermedia of Wrisberg. It enters the superior and anterior compartment at the fundus of the internal auditory meatus. This is the beginning of the aqueductus Fallopii, or Fallopian canal, which passes through the temporal bone, containing the nerve which emerges at the stylomastoid foramen. Its course is at first outward, lying in the angle formed by the superior semicircular canal and the cochlea. As it comes into relation with the internal tympanic wall, it turns backward. This part of the Fallopian canal (tympanic portion) forms a ridge on the internal tympanic wall, which is situated between the oval window and the anterior end of the horizontal semicircular canal. In this part of its course the tympanic wall of the Fallopian canal not infrequently presents defects (dehiscences) which render its involvement in middle-ear processes or its injury during an operation more apt to take place than when these dehiscences are absent. At the posterior tympanic wall the nerve again turns, passing directly downward to the stylomastoid foramen. In this part of its course it lies immediately behind the posterior tympanic wall, at the base of the pyramid containing the stapedius muscle, to which it gives a small branch. In passing through the temporal bone the nerve has thus three directions: the first outward, lying in the coronal plane; the second backward; and the third downward, the second and third being in the sagittal plane. At the junction of the first and second

portions is the geniculate ganglion, which is the origin of the large and small superficial petrosal nerves. These reach the middle fossa through an opening in the Fallopian canal (hiatus Fallopii). The former joins with the large deep petrosal to form the Vidian nerve, which is one of the roots of Meckel's ganglion; while the small superficial petrosal connects with the otic ganglion. These nerves have been considered by some as taste paths, carrying the centripetal impulses received through the chorda tympani. This has been denied by others who believe that the pars intermedia of Wrisberg is the taste path for stimuli reaching the geniculate ganglion through the chorda tympani.

Chorda Tympani.—The chorda tympani is the nerve of taste for the anterior two-thirds of the tongue, being capable of responding to stimuli for the four primary tastes: acid, bitter, sweet, and salty. Its fibers accompany the lingual from which they separate beneath the ramus of the mandible, and passing backward enter a small canal (iter chorda anterious), the canal of Hugier, which runs parallel with the Glaserian fissure, and emerges on the anterior tympanic wall near the anterior margin of the Rivinian segment. The nerve then passes through the tympanum, being enveloped by the anterior and posterior folds of mucous membrane, and lying in the angle formed by the articulation of the malleus and incus. It enters the iter chorda posterious, an osseous canal, which extends from the posterior wall of the tympanum near the posterior margin of the Rivinian segment to the Fallopian canal at the stylomastoid foramen. The chorda joins the facial slightly internal to this foramen and its fibers accompany those of the facial to the geniculate ganglion. In infants that part of the nerve between the tympanum and the facial lies on the surface of the mastoid process beneath the periosteum, merely dipping beneath the upper end of the tympanic ring to enter the tympanic cavity.

INNER EAR.

The best way for the student to acquire a knowledge of the osseous labyrinth is by dissection. The dry bone may be used, but if it is soaked in water for a day or so it will dissect much easier. The instruments used are engravers' tools which may be purchased at the hardware stores at a small expense. The forms which will be found most useful are diamond- and V-shaped. One may by these dissections not only gain a practical knowledge of the canals, cochlea and other parts of the temporal bone, but make some valuable specimens for future study.

The labyrinth occupies a central position in the temporal bone which corresponds to the external part of the petrous portion. It consists of the cochlea anteriorly, the semicircular canals above and posteriorly, and the vestibule uniting these two parts. The soft structures lying within are enclosed by dense ivory-like bone—the labyrinthine capsule. From a physiological standpoint, the labyrinth is divided into two parts, the acoustic and static. The cochlea, being concerned with receiving sonorous vibrations, is called the acoustic labyrinth. The semicircular canals, perhaps also the vestibule, are parts of the apparatus for maintaining equilibrium and are therefore spoken of as the static labyrinth.

Semicircular Canals.—The semicircular canals (Figs. 10–13), three in number, are half-round and consist of a thick ivory-like cortex enclosing a small canal; the diameter of which is about one-twentieth of an inch, except at one extremity where there is an expansion forming the ampulla. This enlargement is for the accommodation of the crista ampullæ, which is a little mound of nerve tissue and an end-organ of one of the branches of the vestibular division of the eighth nerve. Each canal has its ampullated end and its smooth or non-ampullated extremity. An accurate conception of their location and the relative position of their ampullated ends is essential to an understanding of the functional tests of

the static labyrinth. The horizontal or external canal lies approximately in the horizontal plane. Its ampulla

Fig. 10.—Right semicircular canals. From above. For relation of canals to the skull compare with Fig. 14.

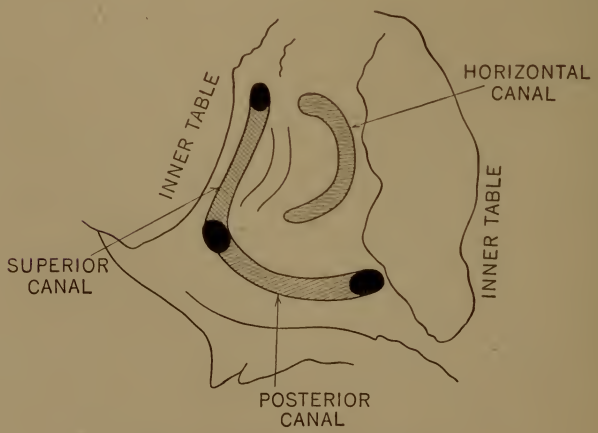

Fig. 11.—Key-plate to Fig. 10.

is anterior and its smooth end enters the vestibule posteriorly. Of all the canals it is the one which is most exposed, as its ivory-like cortex lies bare in the floor of

the antrum. The superior or superovertical canal is, as its name implies, vertical. Its plane is nearly transverse to the axis of the petrous portion of the temporal bone. Its ampulla is in front, below and external, adjacent to that of the horizontal canal. Its smooth end joins with the upper end of the posterior canal to form

Fig. 12.—Left semicircular canals. External view.

a common branch, the crus commune, which opens into the vestibule. Its convexity produces an eminence on the floor of the middle fossa, the eminentia arcuata. In its concavity on the cranial aspect is the subarcuate fossa. In the infant this is a well-marked depression outlining the canal, but in the fully developed bone it is filled to a greater or less extent with osseous tissue.

It nevertheless conveys a vein and a process of dura mater, and through it infection may reach the cranial cavity from the antrum. The posterior or posterior-vertical canal is also vertical. Its plane is nearly parallel with the axis of the bone. Its ampulla is at its lower extremity and its upper end joins with the superior canal to form the crus commune. Its convexity is directed toward the mastoid portion, and in cellular bones may be exposed while performing the mastoid operation.

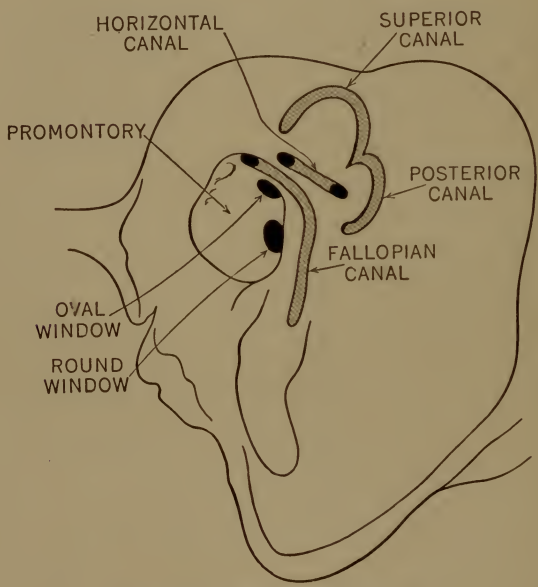

Fig. 13.—Key-plate to Fig. 12.

The relative position of the canals is that the plane in which each lies is practically perpendicular to the plane of the other two (Fig. 14). The axis of the temporal bone forms with the median line (approximately) an angle of 45 degrees, and the axes of the two bones make with each other an angle of about 90 degrees, or a right angle. As the superior canal is transverse to the axis of the bone and vertical, it follows that it must be

in a plane which is parallel to that of the posterior canal on the opposite side, since this canal is vertical and its plane is in the axis of the opposite bone. The horizontal canals on the two sides are in the same plane.

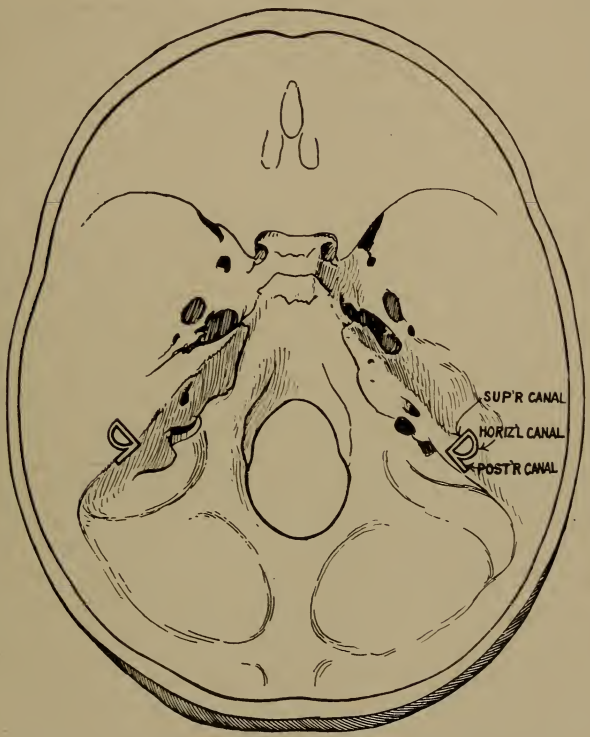

FIG. 14.—Plane of canals and axis of temporal bone.

Vestibule.—The vestibule is a small cavity which lies beneath the bone which exists between the two ends of the horizontal semicircular canal. It varies in size in different bones but the following measurements are correct for an average-sized cavity: "$\frac{1}{5}$ inch from above, downward; $\frac{1}{8}$ inch without, inward" (Gray). It presents the five openings for the three semicircular canals

—above and anteriorly, the ampullated ends of the superior and the horizontal canals; above and posteriorly, the smooth end of the horizontal canal and the crus commune; below and posteriorly, the ampullated extremity of the posterior canal. Besides these five openings are the foramina for the vestibular nerve situated on the inner wall of the vestibule. On its posterior wall is the opening of the aqueductus vestibuli, which is a small canal opening on the posterior surface of the petrous portion between the internal auditory meatus and the groove for the lateral sinus. In the recent state this contains the ductus endolymphaticus, a part of the membranous labyrinth. On the external wall of the vestibule is the oval window, anterior and inferior to which exists the opening into the first turn of the cochlea, containing the canalis reuniens Hensenii which unites the cochlear duct with the saccule. On the inner wall of the vestibule are two depressions separated by a crest-like process. The anterior is round (spherical recess) and lodges the saccule, a small membranous sac which communicates with the cochlear duct. The posterior is oval (elliptical recess) and contains the utricle, a small oval sac, which is connected by five openings with the membranous semicircular canals. From the utricle arises a tube which, joining with a similar one from the saccule, forms the ductus endolymphaticus which extends through the aqueductus vestibuli into the dura on the posterior surface of the temporal bone and is concerned in the regulation of the intralabyrinthine pressure.

Cochlea.—The cochlea, as its name indicates, is shaped like a snail shell. It is composed in the human species of two and one-half turns around a central stem or modiolus. The base of the modiolus rests upon the anteroinferior division of the fundus of the internal auditory meatus and its direction, and therefore the axis of the cochlea, is forward and outward. The beginning of the first turn of the cochlea around the modiolus forms the promontory on the internal tympanic wall. This may be

plainly seen if the drum membrane is removed or destroyed by disease. The second turn lies immediately anterior to the first but forms no prominence. It may, however, be easily opened by the surgeon if thought necessary. The third, which is only a half-turn, is buried in the mass of bone which forms the carotid canal. The modiolus is a fragile, porous pyramid through which the cochlear branch of the eighth nerve reaches the organ of Corti. Meningeal sheaths extend around these nerves into the modiolus so that its fracture during the labyrinth operation may result in the escape of cerebrospinal fluid (Richards). Projecting into the cavity of the cochlea from the sides of the modiolus is a spiral process, the lamina spiralis, which gives passage to the terminal branches of the cochlear nerve. At the base of this lamina is a spiral canal (Rosenthal's canal), which contains a gangliaform enlargement of the cochlear nerve called the spiral ganglion. The lamina spiralis as well as Rosenthal's canal containing the spiral ganglion wind around the axis of the cochlea two and a half times. The free margin of the lamina presents two lips. To the one toward the base of the cochlea is attached the basilar membrane which extends across the cochlear tube and is attached to a thickened part of the periosteal membrane (ligamentum spiralis) lining the outer wall of the cochlea. The basilar membrane extends in a spiral manner from the base to the apex of the cochlea, and divides this cavity into two tubes. The one toward the apex is still further divided by Reisner's membrane, which extends from the surface of the lamina spiralis to the outer wall of the cochlea where it is inserted at some distance from the basilar membrane (Fig. 15). There are thus formed three compartments or spiral tubes. The one toward the base of the cochlea is called the scala tympani, as it begins at the round window in relation with the tympanic cavity, from which it is separated by the membrana tympani secondaria. It extends from this location two and one-half times around the cochlear

axis to the apex or cupola. In this scalæ near the base is the opening of the aqueductus cochleæ by which communication is established with the meningeal cavity. The tube which lies toward the apex is called the scala vestibuli, as it begins at the vestibule and extends to the cupola where it joins the scala tympani, the junction being called the helicotrema. Lying between these two

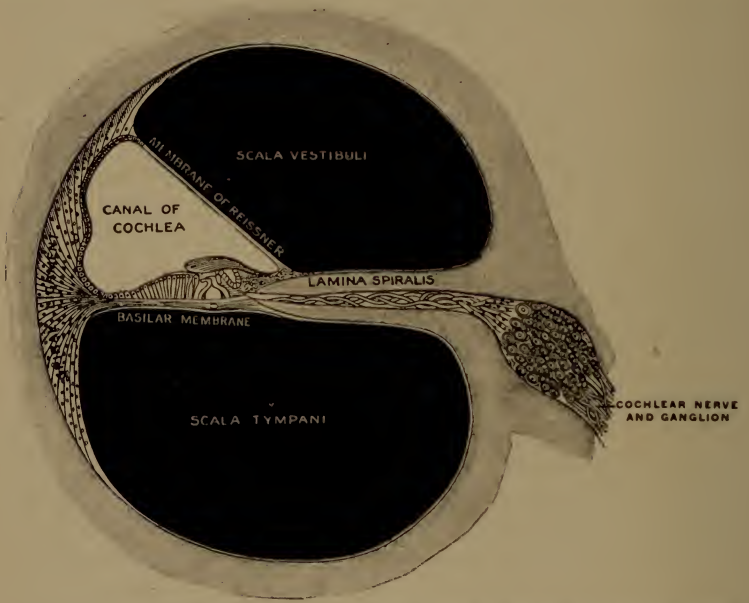

FIG. 15.—Transverse section of a cochlear whorl, showing formation of the scalæ. (Gerrish.)

tubes is the third, the scala media or cochlear duct, which ends in a blind extremity at the apex and at the base opens into the saccule by a small, short canal, the cannalis reuniens Hensenii. These scalæ are filled with fluid; the cochlear duct contains endolymph, while the other two scalæ contain perilymph.

Organ of Corti.—Lying upon the basilar membrane in the scala media is the organ of Corti, which is the end-

organ of the cochlear branch of the eighth nerve (Fig. 16). This organ is formed as follows: There are two rows of supporting cells or rods, an inner (toward the cochlear axis) and an outer row, which join at their apices but are separated at their attachment to the basilar membrane, thus enclosing a spiral space—the tunnel of Corti. They support the hair cells to which terminal filaments of the cochlear nerve have been traced. These hair cells are therefore the real nerve endings of the cochlear branch of the eighth nerve. They are arranged as follows: one row internal to the rods and tunnel of Corti, and several

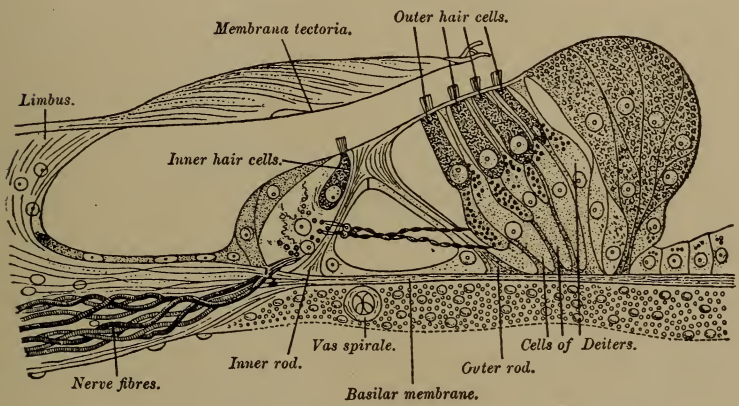

FIG. 16.—Section through organ of Corti. (G. Retzius.)

rows external to these structures. The external hair cells are still further supported by Dieters's cells, which rest upon the basilar membrane by expanded bases, the opposite ends projecting between the hair cells. External to the outer hair cells are the supporting cells of Henson, and still more externally are situated the cells of Claudius. The hair-like processes project from the free ends of the hair cells into the scala media. Stretching over these is the tectorial membrane. In sections of the cochlea this membrane appears variously displaced and distorted but it is probable that in the recent state its periphery

is attached to Corti's organ external to the outer hair cells, and it thus lies floating upon the ciliary processes of the hair cells and by its movement in vibration of the labyrinthine fluid is capable of stimulating these cells, thus producing the sensation of sound.

Blood Supply.—The blood supply of the labyrinth is derived almost exclusively from the internal auditory artery, a branch of the basilar. This divides at the fundus of the internal auditory meatus into the cochlear and vestibular branches. The cochlear divides into small branches which enter the modiolus and pass through canals in the lamina spiralis to supply the membranous structures. The vestibular divides into branches which accompany the nerves, and supply the membranous structures in the semicircular canals and vestibule.

Nerves.—The eighth nerve divides at the fundus of the internal auditory meatus into two parts, the cochlear and vestibular. The latter is posterior and has developed upon it an enlargement, the vestibular ganglion or ganglion of Scarpa. The nerve then divides and enters the foramina in the posterior cribriform areas of the fundus of the meatus (Fig. 17). One branch passes to the crista of the posterior semicircular canal through the foramen singulare. The remaining branches are distributed to the saccule, utricle and also to the cristæ of the horizontal and superior canals. In the cristæ the nerve endings arborize around the hair cells. The cochlear nerve divides into numerous branches which pass through foramina in the antero-inferior division of the fundus and enter the canals in the modiolus. As they pass into the lamina spiralis they enter the spiral ganglion in Rosenthal's canal, emerging from which they pass to the margin of the tympanic lip of the lamina spiralis ossea and entering the organ of Corti are connected with the hair cells.

In tracing the eighth nerve to its central connections, it must be remembered that it is composed of two separate sets of fibers—the cochlear which conduct auditory

impulses, and the vestibular which is concerned with maintaining equilibrium and in orientation. These fibers are in the same trunk form near the fundus of the internal auditory meatus to their entrance into the brain, which takes place in the groove between the pons and the medulla immediately anterior to the restiform body. From this point the two roots have separate connections.

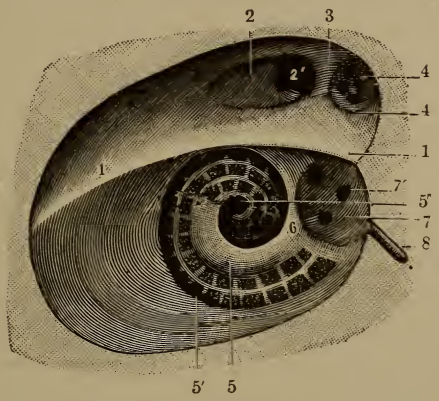

FIG. 17.—Diagrammatic view of the fundus of the internal auditory meatus: *1*, falciform crest; *2*, anterosuperior cribriform area; *2′*, internal opening of the aquaeductus Fallopii; *3*, vertical crest which separates the anterior and posterosuperior cribriform areas; *4*, posterosuperior cribriform area, with (*4′*) openings for nerve filaments; *5*, antero-inferior cribriform area; *5′*, spirally arranged, sieve-like openings for the nerves to the cochlea; *5″*, opening of the central canal of the cochlea; *6*, crest which separates the anterior and postero-inferior cribriform areas; *7*, postero-inferior cribriform area; *7′*, orifices for the branches of the nerve to the saccule; *8*, Foramen singulare of Morgagni, with the anterior portion of the canal which gives passage to the nerve to the posterior semicircular canal. (Testut.)

Vestibular Root.—The vestibular divides into two sets of fibers, one of which connects with the internal nucleus, which is in the floor of the fourth ventricle, while the other set enters the external nucleus which consists of two parts, an inner nucleus (Dieters's) and an outer nucleus (Bechterew's). From this external nucleus, possibly from the internal also, axones pass to the roof nuclei of the

opposite side of the cerebellum. Some fibers of the vestibular nerve also reach these nuclei without entering the external or internal nuclei. Fibers also pass forward from the nuclei of the vestibular nerve to end in the tegmentum, or to arborize around the motor oculi nuclei. Besides these connections fibers from the vestibular nerve pass to a spinal nucleus of undetermined location (Santee).

Cochlear Root.—The fibers of the cochlear nerve enter either the accessory nucleus or the tuberculum acusticum. Axones from these nuclei pass to the superior olive, the trapezoid nucleus, or the lateral fillet, either of the same or the opposite side. From the trapezoid nucleus and the superior olive axones pass to the lateral fillet of the same or opposite side. The lateral fillet, consisting in the main of crossed fibers, is connected with the inferior quadrigeminal body both of the same and the opposite side. Fibers also pass to the internal geniculate bodies and through the acoustic radiation to the cortex of the temporosphenoidal lobe.

Physiology of Audition.—Sonorous vibrations, striking the drum membrane, set this structure into vibration and its movements are transmitted through the ossicular chain and oval window to the perilymph in the scala vestibuli. In order that this fluid may vibrate, some other yielding place must exist in the labyrinthine wall. The round window which is closed by a flexible membrane at the base of the scala tympani, furnishes this resiliency. Thus sonorous impressions set the perilymph in the scala vestibuli and tympani in vibration. This movement is communicated to the flexible tube, the scala media, which lies between them and necessarily affects the organ of Corti which lies in this scala. The vibration produces in this organ a stimulation which is interpreted as sound. Of this there is no question. When it comes to consider in what manner these vibrations act upon Corti's organ to produce the sensation of sound, one enters upon a subject which has given rise to much discussion. The organ of Corti is the structure which interprets the

attribute of sound called pitch. There is no other structure which is at all designed to accomplish this. Pathological processes may result in destroying the ability to hear sounds of a certain pitch. Any theory to be acceptable must furnish an explanation of the manner in which the pitch is recognized, and account for the ability of a pathological process to destroy the pitch perception in a part only of the musical scale. One of the older theories is that of Helmholtz. He believes that the selective action which enables the pitch to be analyzed resides is the basilar membrane. The fibers of this membrane vary in length in a regular manner from the base to the apex of the cochlea, the longer being at the apex and the shorter at the base. He assumes that a fiber, or perhaps a set of fibers, vibrate in consonance with a sound of a certain pitch. The high-pitched sounds, which would presumably carry a shorter distance into the labyrinthine fluid, are assumed to affect the shorter fibers of the basilar membrane near the base of the cochlea, while the sounds of lower pitch which would produce vibrations extending farther toward the apex would cause a vibration of the longer fibers existing in this location. This theory is also a sufficient explanation of the pathological loss of pitch perception in a part of the musical scale. It seems, however, as though nature must have designed a more direct way of stimulating the hair cells than through the basilar membrane, although it is conceivable that vibration of a cell or cells in the basilar membrane could produce motion in a set of hair cells, and excite them by the movement of their ciliary processes in contact with the tectorial membrane. Other theories place the selective action in the rods or hair cells or tectorial membrane. All of these theories are more or less pure speculation.

Labyrinthine Pressure.—The ductus endolymphaticus and the aqueductus cochleæ are concerned in regulating the pressure within the labyrinth. The ductus endolymphaticus is formed by two small tubes which arise,

the one from the saccule, and the other from the utricle. These join, forming this duct, which extends through the aqueductus vestibuli and forms a sac in the layers of the dura on the posterior surface of the temporal bone, between the internal auditory meatus and the lateral sinus. The fluid in this ductus endolymphaticus is the same and continuous with the endolymph within the membranous labyrinth. This sac being compressible and lying mainly within the cranial cavity, maintains the pressure within the membranous labyrinth at that of the cranial cavity. The aqueductus cochleæ, while it does not extend within the cranial cavity, is a direct communication between the perilymph in the scala tympani and the fluid in the subdural space, as it opens into a meningeal process which passes into the posterior lacerated foramen. Colored fluid will find its way from the subdural space into the perilymph of the labyrinth. Thus the pressure, both of the endo- and perilymph, is maintained practically at that of the intracranial cavity.

CHAPTER II.

EXAMINATION OF THE PATIENT.

HISTORY.

BEFORE proceeding to a special aural examination of a patient, his family, personal, and aural history should be obtained.

Family History.—The occurrence of impaired hearing in his parents or several of his relatives may have an important bearing upon the diagnosis. It is a well-known fact that certain aural conditions accompanied by impairment of hearing occur in members of predisposed families. Inherited syphilis may cause aural disease, and if this is suspected, some attempt by indirect questions, may be made to establish the fact that the disease has occurred in the parents. Usually the patient, if this is the cause of his disease, will show the stigmata of this affection.

Personal History.—In a great many instances this is of importance. Many of the suppurations of the middle ear have their inception in the infectious diseases of childhood—typhoid fever, pneumonia, or other general diseases—and this may have considerable influence on the prognosis. So, also, affections of the labyrinth with impairment of hearing may be caused by some of these diseases, as mumps, measles, scarlatina, etc. The occurrence of syphilis, injuries to the head, attacks of dizziness, and many other conditions which cannot be enumerated in detail, all have their bearing in forming an accurate conception of the patient's aural condition.

Aural History.—The aural history is not so likely to be neglected, but nevertheless should be obtained in a thoroughly systematic manner. The cardinal symptoms of ear disease are pain, discharge, impaired hearing,

tinnitus, and vertigo. In ascertaining which of these are present, it is important to inquire as to certain characteristics which these symptoms may present, the significance of which will be developed as the diseases in which they occur are discussed. These characteristics are as follows:

Pain.—The duration, intensity, and location of the pain are to be noted. If pain on pressure, whether it is produced by pressing upon the mastoid process or upon the external auditory canal; whether or not movement of the auricle causes pain. The character of the pain should also be noted, whether continuous and throbbing or intermittent, as in neuralgia.

Discharge.—Concerning the discharge are to be noted the following points: whether profuse or scanty; continuous or intermittent; purulent, sanguinolent, or serous; and total time since it appeared.

Impaired Hearing.—Note the duration since deafness appeared; whether the impairment has been progressive and increased steadily, or intermittently during attacks of nasopharyngeal catarrh or "colds."

Tinnitus.—Tinnitus is a symptom about the presence of which the patient seldom allows the surgeon to be in doubt. It should be put down in the history, but a minute discussion of it with the patient will not lead to any valuable result, as it tends to fix the patient's mind upon this symptom, which is to be avoided as far as possible. During treatment it is to be ignored as far as possible, for if the patient continually directs his mind to this symptom he will endeavor to gauge his improvement by the amount of tinnitus present. This alone tends to exaggerate the subjective noises.

Vertigo.—This symptom when present in aural disease is due to some disturbance of the static labyrinth or vestibular nerve. Its presence or the history of previous attacks may be of great aid in arriving at a conclusion as to the aural disease of the patient. Associated with vertigo one very frequently finds nystagmus. This is an oscillation of the eyes, and may be due to many causes.

ST. LUKE'S HOSPITAL (OUT-PATIENT DEPARTMENT).

NAME
ADDRESS NATIONALITY
OCCUPATION AGE M. S. W. DATE

DEPARTMENT OTOLOGICAL No.
DIAGNOSIS
COMPLICATIONS

Chief complaint		
Impaired hearing	right	left
Tinnitus	"	"
Discharge	"	"
Pain	"	"
Eyes	"	"
Vertigo		
Nausea vomiting		
Headache		
Paralysis		
Nose		
Pharynx		
Larynx		
Course		
Habits		
Past history		
Family history		
Operations		
Genl. health		
Auricle, right		left
Meatus, right		"

R.

L

Mastoid	right	left
Spon. nyst.	"	"
Tubes	"	"
Paralysis		

Watch acoumeter: R. B / A; L. B / A

Moderate low forced. Whisper. Voice: R. B / A; L. B / A

Limits: R. Upper B / A, Lower B / A; L. Upper B / A, Lower B / A

256 bone conduction: R. L.

R. B C A C

L. B C A C

Ataxia—falls to

Turning: Rot to rt. = nystag. to lt. for; " " lt. = " rt. "

Caloric right: Heat / Cold Left: Heat / Cold

FIG. 18.—History chart.

As a rule, when nystagmus arises from ocular or other causes outside of the vestibular apparatus and cerebellum, the movement in each direction is rapid; while in nystagmus arising from disturbance of the static mechanism the movement in one direction is rapid, in the opposite direction slow. Many apparently normal individuals show a slight nystagmus in extreme lateral positions of the eyes. This is considered of no special significance, and is called physiological nystagmus. As the eyes become fixed it usually subsides or becomes less, while in vestibular nystagmus fixation seems to have no effect or intensifies the movements.

In taking the history, as well as in recording the results of the subsequent examination, a history sheet with the headings of the important points to be brought out is of great value. The one reproduced (Fig. 18) will be found to answer practically all requirements.

METHOD OF EXAMINATION.

Illumination.—Probably the best light for aural examination is the incandescent electric candle, the film being either of carbon or tungsten. The globe may be frosted if desired, and with or without a fenestra. A reflector and condensor may be used, but possess no advantage over the plain candle. The light may be attached to a stand with a flexible neck and extensible tube to vary the height (Fig. 19). With this apparatus the light may be placed in any desired position, or the same result may be attained by mounting it upon a wall bracket. An Argand gas burner attached to the wall by a bracket moving in any direction gives very satisfactory illumination. A condensor and reflector may be used if desired. In emergencies outside of the office one may use an ordinary coal oil lamp with Argand burner, or even a candle when other sources of illumination are unavailable. If the examiner is accustomed to the electric light and uses some of the other forms of illumination he should make

proper allowance for the resulting change in color of the parts, as they will not appear so white. Numerous forms of apparatus have been devised, in which the electric candle is worn on the examiner's head, the current being obtained from a battery or from the regular house current.

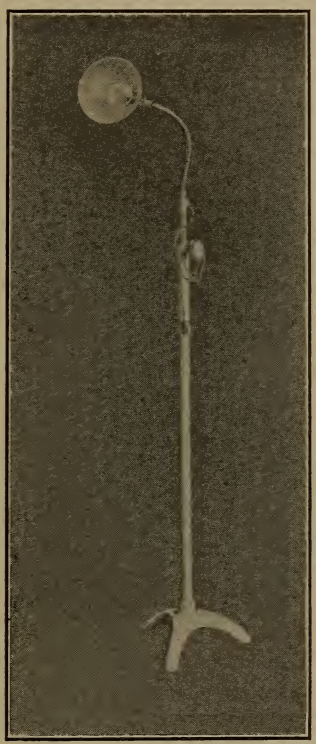

Fig. 19.—Electric light stand.

The light from these is very useful in operations, but they do not give that definition which is essential to obtain a satisfactory view of the membrana tympani.

Mirror.—In selecting a forehead mirror certain points should be borne in mind. Three to three and one-half inches is the proper diameter. A larger one is more

difficult to manage and tiresome to wear and presents no points of advantage. The central aperture should be from one-half to five-eighths inch in diameter. Proficiency in the use of the mirror, if one with a small opening is used, can only be acquired after much pains and time, and even then it will be found much less convenient for every-day work. The ball should be attached to the periphery of the frame (Fig. 20), thus allowing much more freedom of motion than if attached to the back. The mirror is connected with the headband by a double ball-

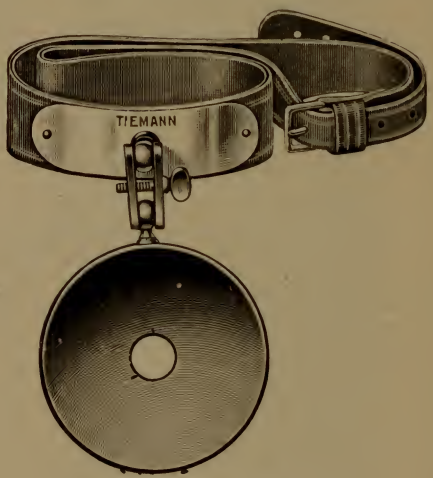

Fig. 20.—Forehead mirror.

and-socket joint. If some object, for instance the window, is picked up by the reflecting surface of the mirror it should produce a clear, well-defined image of the same, showing accuracy of curvature. This ought to be about nine inches from the mirror, as this length of focus has been found to be the best. To use the forehead mirror it is necessary to train the muscles of the neck and body, so that whatever movements are executed by the hands the head remains fixed and the parts under observation continue illuminated. To obtain this training rapidly

the following procedure is advised: With the mirror in position, let the student pick up the light and focus it upon the wall, taking care that the illuminated area is seen through the aperture by the eye behind the mirror. Then let him make as extensive movements as possible with his hands and arms, at the same time holding the light fixed in the original position. By perseverance in this exercise the student will soon overcome his difficulty with the illumination. The light should be on the examiner's left and so placed that the head and face of the patient on the side to be examined just fall in shadow.

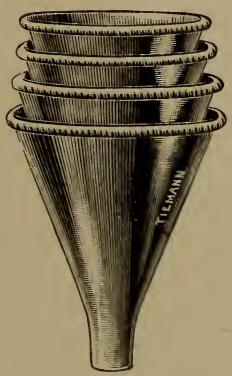

FIG. 21.—Aural specula. (Boucherons.)

Introducing the Speculum.—Before introducing the speculum the condition of the auricle and the size of the meatus is noted. If the meatus is contracted or manipulation of the auricle is painful, extraordinary care in introducing the speculum must be taken or pain will be produced. The ordinary bell-shaped speculum is recommended (Fig. 21). They come in nests containing four sizes. Choose the largest size that can be introduced without using the slightest force. This is inserted into the meatus and the parts inspected.

Meatus.—Three points are to be noted about the external auditory canal, its color, caliber, and contents.

Color.—The normal color of the canal wall is about the same as that of the skin. One may find localized areas of reddening in circumscribed inflammation or diffuse, as in eczema and diffuse otitis externa.

Caliber.—The caliber of the entrance of the meatus has already been noted before introducing the speculum, but now the deeper part of the canal is to be investigated. Whether it is narrow at the fundus, as in mastoiditis, or in the soft part of the canal, as in furuncle.

Whether the thickening of the walls is general, as in diffuse otitis externa, or limited to the superior or posterior wall, as when pus has dissected its way out, beneath the periosteum, through the Rivinian segment. The consistence of the thickening should be determined; whether it is hard, as in exostoses, or less firm, as in the majority of other processes which diminish the caliber of the canal.

Contents.—The contents may be fluid or solid, of any consistence between these extremes, or the canal may be empty. Pus may be thin and watery, of creamy consistence, or dried into hard scales on the canal walls. Any crust should be looked upon with suspicion which is found near the drum membrane, especially upon the superior wall, as it may be dried pus concealing a perforation which leads into the tympanic vault. All of the characteristics of the pus are to be noted as it is removed with the cotton-wrapped applicator: its consistency, amount, color, and odor. It may be necessary in nervous children to remove it with the syringe before a view of the fundus can be obtained. Sometimes the discharge is sanguinolent, a condition which may exist with polypus, granulations, hemorrhagic otitis externa or following traumatism. Nor can one be satisfied in many instances until the aural pus is submitted to a microscopical examination of the smear or culture. In taking the pus for a smear or culture, it is important to secure it fresh from the middle ear. In order to do this the canal is first wiped dry. Then the ear is inflated by Valsalva's method if possible, if not, with the catheter. This forces pus from

the middle ear into the canal and it is taken on an applicator. If a smear is to be made, this is thinly spread on cover-glasses and the film fixed by rapidly passing them through a flame. They are then stained and examined. If a culture is desired, the sterile applicator is used and the pus is placed on slants of the proper culture media. Cerumen is usually found in small amount and of soft consistency in the external part of the canal, but often it will be found occluding the canal lumen. This is to be removed and the examination continued. The canal may contain foreign bodies. Those most frequently found when the patient is ignorant of their presence are wads of cotton which he has inserted at some previous time under the mistaken notion that they were some protection to his

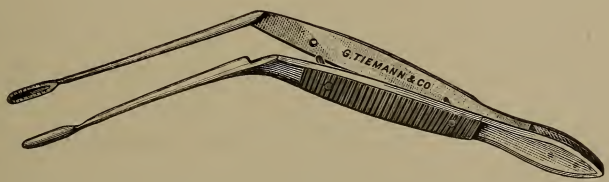

Fig. 22.—Angular forceps.

ears. For removing these, as well as epithelial scales, masses of fungi, etc., the angular forceps (Fig. 22) are of value.

Membrana Tympani.—Having cleared the canal of its contents, if any were present, the deeper parts are to be inspected. In order to obtain a good view of the fundus, one anatomical point must be borne in mind. The external auditory canal consists of two parts; the outer cartilaginous portion is movable and has a direction upward, backward, and inward; the inner or bony portion is nonmovable, with a direction downward, forward, and inward. By traction upon the auricle in a direction upward and backward, these parts are brought into the same line and a view of the membrana tympani obtained. In young children the canal is straightened by drawing the auricle

backward and downward. If the student experiences difficulty in locating the drum membrane, it is a good plan to follow with the eye one wall of the canal to and across the fundus and back to the speculum on the opposite wall. In this procedure one necessarily passes over the drum membrane if it is present. The different points to be noted are: (1) short process of the malleus; (2) handle of the malleus; (3) Shrapnell's membrane; (4) membrana tensa; (5) cone of light; (6) anterior and posterior folds (Fig. 23). The hammer handle (2) or manubrium will be seen as a yellow streak extending from about the centre of the tympanic membrane in an upward direction

Fig. 23.—Diagram of left membrana tympani: *1*, short process of the malleus; *2*, manubrium or handle of the malleus; *3*, Shrapnell's membrane, or membrana flaccida; *4*, membrana tensa; *5*, cone of light; *6*, anterior and (*6'*) posterior folds.

and ending in a small spike-like process, the processus brevis or short process of the malleus (1). Extending from this in either direction are the anterior and posterior folds (6). These correspond, in position, more or less closely to the folds of mucous membrane in the tympanum already described, and divide the membrane into two parts; the upper being Shrapnell's membrane, or membrana flaccida, and the lower, the membrana tensa. Shrapnell's membrane is of great importance since it is the only part of the wall of the vault, or upper part of the tympanic cavity, which can be directly inspected. It has in the normal state about the appearance of skin. It is of a delicate pink color, without luster as compared

with the membrana tensa. It is continuous with and of the same appearance as the superior canal wall. Owing to the obliquity of the anterior and posterior folds, and since it fills in the segment of a circle, the vertical measurement of Shrapnell's membrane is greatest from the short process upward, diminishing to its anterior and posterior extremities which lie at the ends of the tympanic ring. Normally, it is slightly concave. It should be carefully examined for perforations, bulging or redness, although the color may vary considerably without pathological significance.

Quadrants of the Membrana Tensa.—For convenience of description, the membrana tensa is divided into quadrants

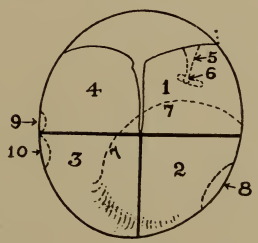

FIG. 24.—Diagram of the quadrants of the tympanic membrane: *1*, posterosuperior of hearing quadrant overlying incus (*5*) and stapes (*6*); *2*, postero-inferior quadrant over niche of round window (*8*); outline of promontory (*7*); *3*, antero-inferior quadrant; *4*, anterosuperior quadrant; tympanic opening of canal for tensor tympani (*9*) and Eustachian tube (*10*).

by imaginary lines (Fig. 24); one extending from the tip of the manubrium to the floor, and another at a right angle to this at the tip of the manubrium or umbo, the hammer handle completing the division. A knowledge of the tympanic structures which underlie these quadrants is essential. The posterosuperior quadrant lies over the oval window containing the stapes articulated with the long process of the incus. For this reason it is sometimes spoken of as the hearing quadrant. These structures may not infrequently be seen when the membrana tensa is destroyed, or for any reason is thin and transparent. At the posterior margin of the postero-inferior quadrant

is the niche of the round window, the margin of which may sometimes be seen when the tympanic membrane is absent. The parts of the posterior quadrants toward the umbo or end of the manubrium overlie the promontory. In this location the drum membrane approaches nearer to the internal tympanic wall than at any other place, in a normal tympanum these structures being about one-twelfth inch apart. Opposite the anterior quadrants the internal tympanic wall is receding until near the tympanic ring it attains its maximum distance from the membrane, which is about one-fifth of an inch. There is, however, considerable variation in both of the measurements, above given, in the tympani of different subjects. At the margin of the anterior segments and at a slightly deeper level is the tympanic opening of the Eustachian tube, and above it the opening of the canal for the tensor tympani muscle. In the recent state the canal is occupied by this muscle, and its tendon crosses the tympanum to its attachment to the manubrium. Of the membrana tensa (Fig. 23, *4*) there is to be noted the color, luster, position, integrity, and structure.

Color.—The only way the student can form an accurate conception of the normal color and luster of the tympanic membrane is by making repeated examinations of normal ears. This he should do so that he may be quick to perceive an abnormal condition when it presents. The normal color of the drum membrane is a grayish tint, but it varies considerably within physiological limits. If the membrane is very thin the color of the mucous membrane on the internal tympanic wall may show through. This pink color must not be attributed to the drum head to which it does not belong. The tympanic membrane becomes of varying degrees of redness in inflammatory diseases of the middle ear. Care should be taken not to misinterpret the physiological redness along the hammer handle and around the circumference of the drum membrane. This is due to injection of the manubrial and peripheral plexuses, and frequently occurs after prolonged

examination with the speculum inserted or after removal of the cerumen and is without special clinical significance.

Luster.—The normal luster is a quality in itself. It is a peculiar sheen not very unlike mother of pearl, and is due to the character of the epithelium which covers the drum membrane. When for any reason this becomes necrotic or exfoliates *en masse*, the drum head becomes lusterless. This may occur in certain forms of otitis externa, but is a much more important indication of acute purulent otitis media, and in this condition the membrane at times very much resembles wet blotting paper. Besides this total loss of luster, varying degrees of its diminution may be present. Care should be taken not to confuse the cone of light with luster. This may be absent, as it is in many instances in perfectly good ears, and the drum membrane be of normal luster. The light reflex is caused by the concavity of the anterior part of the membrane which allows rays of light to strike the area of the reflex perpendicularly and be reflected back to the eye. The normal obliquity of the membrane is such that rays of light from the forehead mirror are reflected downward, forward and outward, to the anterior wall of the canal. Contraction of the tensor tympani depresses the manubrium and consequently the drum head along this process, the depression being greatest near the umbo or end of the manubrium. This increases the obliquity of the upper and posterior part of the membranes and diminishes that of the lower and anterior part. Thus an area is formed which is nearly perpendicular to the axis of the canal, striking which the rays of light from the mirror are reflected back to the eye. So while a good luster must be present in order to produce a well-developed cone of light, its absence does not, in itself, indicate a loss of luster. A well-formed light reflex begins at the umbo and spreads out in a triangular form to near the anterior and inferior margin of the tympanic ring.

Position.—If the depression of the drum membrane becomes extreme the light reflex recedes from the umbo

and loses its cone-like shape. In forming an opinion as to the amount of depression of the drum membrane much more reliance should be placed upon other indications. These are: (1) Foreshortening of the manubrium; as this structure is viewed more obliquely it appears shorter; (2) prominence of the posterior fold; (3) prominence of the tympanic ring; (4) apparent narrowing of the posterior part of the membrane. Bulging is usually quite easily made out. The appearances which exist are as follows: (1) broadening of the posterior segments of the membrane; (2) apparent lengthening of the manubrium, which at times seems to lie in a gutter; (3) the posterior fold is more or less obliterated; (4) the tympanic ring is less prominent and may even appear depressed.

Integrity.—Some perforations are readily made out. If difficulty is experienced auto-inflation or catheterization may aid one. The pus is wiped away so that a good view of the tympanic membrane is afforded then inflation is performed (usually by the patient) and the location at which pus first appears is noted; this of course will be the site of the perforation. It may be impossible to force the contents of the middle ear through the perforation either by auto-inflation or by catheterization. This may occur in posterior perforations where the anterior part of the cavity is closed off by adhesions or the cicatricial drum membrane is adherent to the promontory; or in perforations in Shrapnell's membrane, where, as may be the case, the vault is separated from the lower part of the tympanic cavity by the thickened folds of the mucous membrane. In this event a Siegel's otoscope may be of value (Fig. 25). This instrument is so constructed that the air pressure may be increased or diminished in the external meatus, the examiner in the meantime having a view of the fundus of the canal. By producing a minus pressure any secretion which may be in the tympanum will be drawn out. By alternately condensing and rarefying the air in the meatus a very good idea of the amount of motility of the drum membrane may be obtained.

Inflation of the middle ear when a perforation is present may also give a certain amount of information as to the size of the opening. After the tympanic contents are forced out and air comes through, the pitch of the perforation sound will be high in small openings and low in larger perforations. To a certain extent the size of the perforation is an indication as to whether or not drainage is good. With a high-pitched whistle showing a small perforation, drainage is presumably insufficient, while with a low-pitched sound produced by a large perforation, drainage, at least as far as the membrana tympani is concerned, may be considered satisfactory. In total absence of the membrana tympani, the internal tympanic wall may be

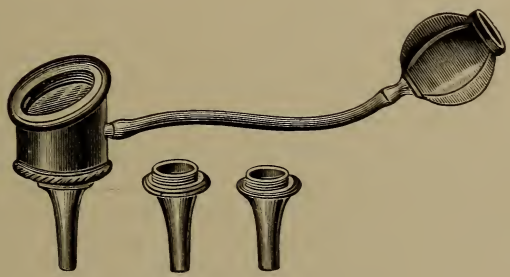

FIG. 25.—Siegel's otoscope.

mistaken for a red bulging drum membrane. Tracing along the wall of the canal to and over the fundus, will frequently enable one to avoid this error. It will be found that as one's vision passes over the tympanic ring to the internal tympanic wall, a well-marked offset exists. Inflation and the use of the probe are also suggested in case difficulty in identifying the nature of the structure is experienced.

Structure.—The thickness and translucency of the membrana tensa varies greatly. Some membranes are nearly transparent and allow the stapes and long process of the incus to be seen and the pink color of the mucous membrane on the internal tympanic wall to show through.

Others are nearly or quite opaque. Thin atrophied membranes are at times associated with chronic catarrhal processes. Opacity of the drum head may be due to the deposit of lime salts or to thickening the result of acute or chronic purulent inflammation of the middle ear.

It is a good plan to examine the opposite ear before the involved one, as a comparison can then be made. In any event it should always be examined. The condition of the nose and nasopharynx should also be investigated as a matter of routine.

INFLATION OF THE MIDDLE EAR.

Inflation of the middle ear is at times a part of the examination, at others it is a therapeutic procedure. It consists in forcing air under pressure into the middle ear and may be accomplished in three ways: by Valsalva's method, by Politzer's method or by catheterization.

Valsalva's Method.—In this method the inflation is performed by the patient. It is therefore frequently spoken of as auto-inflation. The patient closes the nose by the thumb and finger so that no air can escape, and at the same time endeavors to force air through the nose. This produces a positive pressure in the nasopharynx and the air is forced through the Eustachian tube into the middle ear. Valsalva's method may be used to assist in finding a perforation of the drum membrane, and to give information as to the amount of drainage in middle-ear suppuration. If the air blows through the ear freely, one may estimate the degree of drainage present. If it is not forced through at all, no evidence as to the drainage is furnished, as the force exerted in this manner may be insufficient to overcome the resistance offered by a swollen or contracted tube. For all other purposes Valsalva's method is to be condemned.

Politzerization.—In Politzerization the positive pressure in the nasopharynx is produced by compressing a rubber

bulb which is connected by a tube with a tip of proper size and shape to occlude one nostril, the other being closed by the operator. The nasopharynx is closed off below by the elevation of the soft palate until it comes in contact with the posterior wall of the pharynx. This is accomplished by having the patient take a swallow of water, utter the sound represented by "K," or by performing the act of blowing with the mouth closed. As the sound is uttered, or the patient swallows, the bulb is compressed and the air enters the tympanum through the Eustachian tube. If the auscultation tube is used, a sound will be heard as the tympanum fills with air. This procedure may be used in children who will not submit to catheterization, or in adults if for any reason it is impossible or seems inadvisable to catheterize them. It is much inferior to this latter procedure both in its therapeutic effect and the information as to the state of the tube and middle ear which it affords.

Catheterization.—*Instruments.*—The instruments needed for catheterization are an auscultation tube, catheters, and an inflating bag with its connections. The auculta-tion tube is two and one-half to three feet in length and one-fourth inch in diameter. Ordinary red rubber is the best material. The pure gum black-rubber tubes are too elastic and for this reason do not give one as good a sound as the less resilient tube. The white or operator's tip should be large enough to plug the canal tightly, and the patient's, or black one, should not be too small, as upon the formation of an air-tight connection between the meatus of the operator and that of the patient the proper appreciation of the inflation sound depends. The most useful catheter is a No. 2, although for exceptional cases either a No. 1 or a No. 3 will afford better results (Fig. 26). The total length of the catheter is six inches. If a longer one is used, it will interfere with the delicacy of the manipulation; if a shorter one, it will not reach to the Eustachian orifice in some patients. The material may be German silver, nickel-plated or coin silver. The latter has the

advantage that the curve may be more easily changed to suit individual requirements. The tip may be either plain or slightly bulbous. The ring shows the direction of the tip, and is of use while holding the catheter in position during compression of the bulb. The Dench bag and apparatus is advised (Fig. 27). This consists of a rubber bulb, a hard-rubber bottle, and a tube about two feet long connecting with the catheter by a hard-

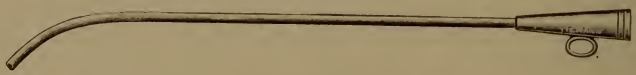

Fig. 26.—Eustachian catheter.

rubber tip. In selecting the bulb one is to be chosen that does not leak at the valve and fills quickly. The bottle is so constructed that the air forced through the catheter may be impregnated with vapor if desired. If the rubber tube connecting the bottle with the catheter is very elastic, too much force will be lost in transmission, if at all rigid it interferes with the delicacy of manipulation in passing the catheter.

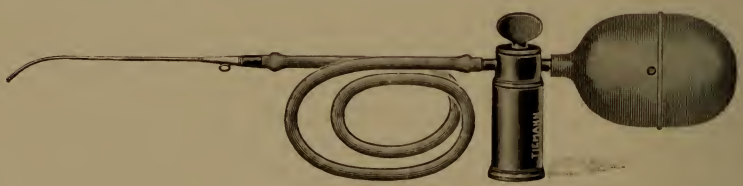

Fig. 27.—Dench inflating apparatus.

Anatomy.—Some anatomical points must be constantly borne in mind during the passage of the catheter. The pharyngeal orifice of the Eustachian tube, into which it will be necessary to insert the catheter, is on the outer wall of the nasopharynx slightly above the plane of the hard palate. There is considerable variation in the location of this orifice. Judging from experience in passing the catheter, the usual position seems to be a little less

than one-half inch anterior to the posterior wall of the nasopharynx, and about one-fourth inch above the plane of the floor of the nose. The posterior lip of the tubal orifice is cartilaginous and is an important guide in inserting the catheter. It varies greatly in prominence in different subjects. The tube passes in a direction outward, upward, and backward. Before attempting to pass the catheter the student should examine the nose, especially noting: the roominess of the inferior meatus; the prominence and position of the inferior turbinate; the presence of spurs or deflections of the septum. He will then be able to determine what modification of the procedure may be necessary to insure success.

Anesthesia.—Anesthesia of the mucous membrane of the inferior meatus and tubal orifice is advisable, especially in the first inflations attempted by the student. Spraying the nose with a 4 per cent. solution of cocain, or some other anesthetic, then with a cotton-wrapped applicator swabbing the tubal orifice with some anesthetic solution will properly benumb the route over which the catheter is to be passed. If the applicator is bent at the end to a curve resembling that of the catheter, it may be passed in the same manner as the catheter, directly into the tubal orifice. That it is in this position may be determined by the fact that a semi-fixed sensation is communicated to the hand. In this way a certain amount of information as to the location of the tube will be obtained that may be of value.

Passing the Catheter.—The patient sits facing the operator with the chin well down toward the chest. This enables one to pass the catheter through the lower meatus with more certainty and ease. The fingers of the left hand rest upon the bridge of the nose, while with the tip of the thumb the end of the nose is tilted sharply upward. The thumb must not interfere with the lumen of the nostril. If these directions are not observed, the internal end of the catheter is in danger, either of entering the middle meatus, thereby causing pain and failure, or of

reaching the nasopharynx so high that when rotated into position it fails to enter the orifice of the tube. At first the student will do well to let the patient hold the bottle or bag until the catheter is in position. The catheter is to be attached to the tip in such a manner that when the rubber tube is at rest, the ring will point toward the ear to be inflated. If the connecting tube is twisted

Fig. 28.—First step of introduction of the catheter. Tip of the nose held up, the right hand depressed. The point of the catheter has entered the vestibule and the direction and extent of the movement of the right hand now taking place is indicated by the arrow.

while search is being made for the tubal orifice, the resistance communicated to the fingers will be sufficient to mask the sensation which ought to be perceived when the tip of the catheter comes in contact with the posterior lip of the tubal orifice. The catheter or the tubing near it is lightly grasped by the thumb and fingers of the right hand exactly like a pen. If grasped firmly, pain will be

caused during the passage of the catheter and failure is almost certain. The tip end of the catheter is now to be inserted into the nose, point downward, with the right hand depressed (Fig. 28). As the tip passes the vestibule, the right hand is raised until the shaft of the catheter comes nearly in contact with the tip of the nose which is still held up by the thumb of the left hand. It is now passed through the lower meatus with tip of the catheter in contact with the floor of the nose. Absolutely no force is to be used in this part of the procedure. If necessary for it to pass freely the catheter may be allowed to rotate. As soon as the tip of the catheter lightly touches the posterior pharyngeal wall it is allowed to rotate, the elasticity of the rubber tube accomplishing this. The ring of the catheter and therefore the tip now point externally. As this rotation is taking place the catheter is slightly withdrawn and the external end pressed against the septum. This combined movement results in bringing the tip of the catheter in contact with the external pharyngeal wall posterior to the tubal orifice, either at the posterior lip of the tube or slightly behind this point. With this contact maintained by gentle pressure against the septum with the outer end of the catheter, it is now drawn forward over the posterior lip into the tubal orifice (Fig. 29). That it is in position here will be recognized by a semifixed feeling communicated to the right hand. The ring of the catheter should point in the direction of the ear to be inflated. The left hand now grasps the catheter, the ring lying between the thumb below and the forefinger above, the other fingers still maintaining their rest upon the bridge of the nose. The right hand thus freed compresses the bulb and if all has gone well the operator will perceive by the sound heard through the auscultation tube that he has succeeded (Fig. 30). If he has not, no attempt should be made to search for the tube with the left hand, but the hands are to be replaced and with the catheter in the fingers of the right hand search made for the location which gives the sensation of semifixation.

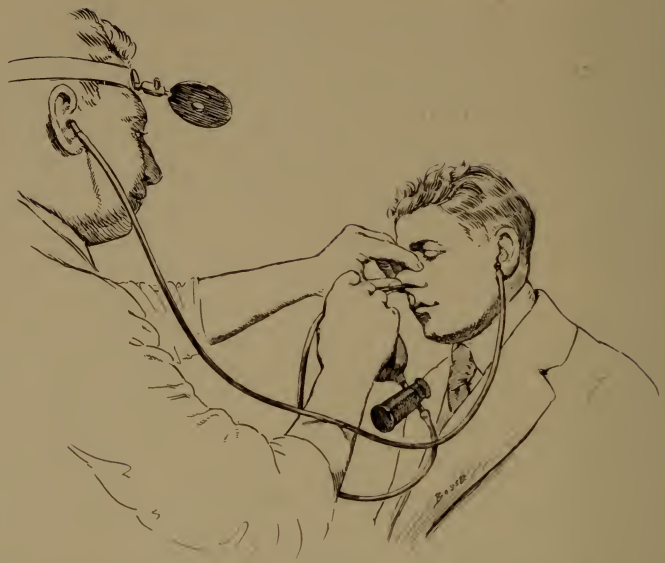

FIG. 29.—The point of the catheter has passed through the lower meatus and is now in the region of the tube for which search is being made.

FIG. 30.—Catheter in the tubal orifice, held in place by the left hand while the bag is being compressed by the right hand.

Causes of Failure.—The causes of failure are generally that the above rules have not been carried out exactly or that the hand and fingers are not sufficiently educated to interpret the sensations communicated to them. This skill will come with practice but the directions above given must be thoroughly understood before attempting catheterization. At times failure results from anatomical conditions. If the lower turbinate is very large and the nose is roomy the curve of the catheter may be increased to enable the tip of the catheter to reach the Eustachian orifice. If the nose is narrow and the turbinate prominent, a very short curve may be used and the catheter passed outside of the turbinate, the tip of the catheter traveling along the external and superior angle (naso-antral wall) of the inferior meatus. It is introduced by sight external to the lower turbinate and passed backward until it meets with the anterior surface of the posterior lip of the tube, when it will be found to be in the tubal orifice. This same method may be tried if the septum is so prominent that it is impossible to pass the catheter between it and the lower turbinate. In some deflections bending the catheter to conform to the septal deformity will enable one to succeed. With an anterior deflection with but little room an instrument with very small curve may be passed and success achieved. If the deflection of the septum is so extreme that the catheter cannot be passed by any of these maneuvers, either the deformity must be corrected by operation or the opposite side used for the passage of the catheter. This is possible in a great many patients but cannot be regarded as a satisfactory procedure. An instrument of large curve is used and the posterior border of the septum taken as a guide, the Eustachian orifice being about opposite to this landmark; or the catheter may be introduced into the orifice of the tube corresponding to the open side of the nose, then, without changing its depth, rotated to the opposite side, when it will generally enter the tubal orifice.

Other Methods.—Another method of passing the catheter is advocated by some authors. The instrument is passed into the nasopharynx as above described, then the point is rotated to the side opposite to the one which it is proposed to catheterize. It is then withdrawn until the curve of the catheter comes in contact with the posterior border of the septum. The point of the instrument is now made to describe a semicircle, the convexity of which is downward. This movement brings the tip of the catheter into the pharyngeal orifice of the tube which it is desired to catheterize. This method requires much more manipulation of the instrument and the point of the catheter irritates the structures in the nasopharynx and often leads to troublesome retching, so that the catheter is firmly held by the contraction of the palatal muscles and its insertion, or even its withdrawal, may be impossible until this contraction subsides. This rarely occurs in the first method which has been described in detail, and which is very much superior to the one in which the posterior margin of the septum is taken as a guide. By using the posterior lip of the orifice of the tube as a guide, and following closely the directions given above, the student will not only be able as readily to learn to inflate as by any other method, but he will also lay the foundation for the acquisition of great skill in the use of the catheter.

Interpretation of Sounds.—All sounds of intratympanic origin are heard through the auscultation tube as though arising in one's own ear. All sounds which seem to come from outside the examiner's ear are either tubal or produced at the mouth of the tube. The sound usually first heard is due to the vibration of the drum membrane, and is more or less pronounced as the membrane is tense or flaccid, varying from a slight thud to a sharp snap or click. Then follows the murmur caused by air rushing through the tympanum. The pitch of this murmur varies with the patency of the Eustachian tube. The narrower the tube, the higher the pitch and *vice versa*. Fluid in

the middle ear produces either crackling or bubbling sounds. If there is a perforation in the drum membrane of small size there will be a whistle; if of large size the operator will feel the air blowing in his own ear. Sounds produced at the mouth of the tube are at times very annoying to the inflator, and are generally caused by the vibration of the mucous membrane; either because it is relaxed and flabby or because the catheter is not directed in the axis of the tube. One may usually get rid of them by rotating the catheter slightly, by gentle traction or pressure; or a catheter of different curve may be tried. These procedures are intended to eliminate the little fold of mucous membrane, vibration of which causes the noise, and they may also be adopted to produce a direct blast of air through the tube into the middle ear, when for any reason the one which is passing is not satisfactory.

Accidents of Catheterization.—Catheterization may be considered as a very safe procedure. Very rarely an emphysema is produced by entrance of air through an injury caused by the end of the catheter or a rupture of the mucous membrane lining the tube. As this submucous emphysema occurs the patient suddenly becomes extremely anxious and the operator may have noticed a puffiness of the check or neck. He must desist at once and not attempt another catheterization for several days. If one examines the throat in these patients the uvula and soft palate will be found swollen from emphysematous infiltration. The accident is free from danger if the hand bulb is used, but with compressed air with continuous flow the emphysema may extend to the glottis, causing distressing dyspnea. The condition heals without treatment. If compressed air has been used and dyspnea is present, a rapid tracheotomy would be indicated. These severe types are so rare that but a few cases are on record. Another accident which may occur is rupture of the membrana tympani. This usually occurs in cicatricial membranes and is quite startling to the operator as well as to the patient on account of the pop which is heard by both.

This accident is by no means common and may be unavoidable, but in a certain type of case it may be prevented, as one has timely warning. The operator hears one inflation sound, but upon further compression of the bulb no sound is produced. If he will stop now and examine the membrana tympani, he will find it markedly bulging. If he persists in trying to obtain another inflation sound, the membrane will rupture. The explanation is that the tube is so narrow that the air cannot escape after the tympanum is filled, so, as more air is forced in, no further sound is produced but the membrane ruptures. These perforations usually heal without treatment and with no noticeable loss of function.

FUNCTIONAL TESTS OF AUDITION.

In testing the acoustic function of the ear, two kinds of tests are made use of: (1) those which furnish data as to the degree of impairment of hearing; (2) those showing the kind of impairment. The first are designed to ascertain and enable one to keep a record of the amount of deafness; while the second indicate with more or less accuracy the location of the lesion causing the impairment of hearing. The amount of deafness is usually determined by the watch or acumeter, and the human voice either in the form of a whisper or a spoken voice. For ascertaining the kind of deafness, the upper and lower tone limits are determined and certain tests made with tuning-forks.

Watch.—The distance at which the watch is heard by the ear being examined is ascertained and set down while beneath it is placed the distance at which the watch is heard by the normal ear. For example: R. E.: watch, $\frac{5}{20}$, denotes that a watch is heard at five inches, which the normal ear hears at twenty inches. There is so much variation in the loudness of watches that simply to mate that a patient hears the watch at a certain distance conveys no accurate impression of the amount of hearing which he possesses; so the number under the line is

added to denote the distance at which the normal ear hears the watch used in the test, thus indicating the loudness of that particular watch. The symbol is not in any way to be considered as a fraction which represents the hearing power. A patient whose hearing for the watch is expressed as $\frac{5}{20}$ has not lost three-fourths of his hearing any more than one who cannot hear the watch, written $\frac{0}{20}$, is totally deaf. When the watch is heard on contact it is written $\frac{C}{20}$. If these considerations are borne in mind, the formula in the form of a fraction is useful in recording the amount of hearing for the watch.

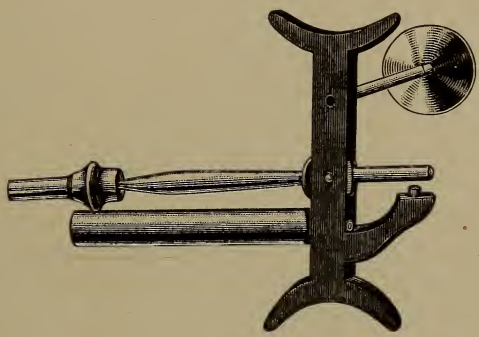

FIG. 31.—Politzer's acumeter.

Acumeter.—When the watch cannot be heard, Politzer's acumeter may be used (Fig. 31). The sound, from this instrument, is produced by the hammer striking the metal cylinder, as it falls after being raised by the finger. This produces a clicking sound of high pitch which is heard by the normal ear under favorable conditions at a distance of 45 feet. In using this instrument care must be taken that the hammer falls with its own weight only, after being raised by pressure on its short arm, for if it is caused to strike with force, a louder sound will be produced and the test be inaccurate. In testing the hearing with this instrument it is started outside the range of audition and gradually brought nearer until the patient hears it. The distance in feet at which this occurs may be set down above

the line with 45 below as in the watch test, or merely noted in feet or inches, as the case may be. Both the watch and the acumeter produce a high-pitched sound of peculiar quality, which it is not very important for the ordinary person to hear. The impairment of hearing which leads most patients to seek advice is their inability to hear the human voice; so this becomes a much more important means of testing the hearing power. Some patients will hear the watch and acumeter much better than the voice; others again will hear the voice much better than these instruments. The watch is valuable in detecting slight degrees of impairment in patients in whom no noticeable deafness for the whisper or voice is present.

Whisper.—In using the whisper to determine the acuteness of hearing, its loudness must be taken into consideration. It is customary to employ three degrees of intensity in the whispered voice: the low, the moderate, and the forced whisper. The low and the forced whisper have the least variation in their degree of loudness. The numbers from 20 to 100 are usually spoken and are the best, as they do not allow the patient to guess at any part of what is spoken, as he is able to do when he hears a sentence. For this reason, the examiner should not allow himself to use a regular sequence of numbers, as deaf patients are very keen in anticipating the ones to be spoken if this is done. The patient closes the opposite ear with the moistened finger and must not be permitted to see the speaker's lips. As the examiner moves away he comes to a place where it is evident that the patient hears the sound but cannot distinguish the numbers. The distance at which this occurs is usually put down as the hearing distance. Thus, R. E.: Low, moderate, forced, whisper = 5 feet, denotes that the whisper is heard at five feet and that it is the low, moderate or forced whisper, as the case may be, a line being drawn through each of the other two. If the examiner starts beyond the range of audition and gradually approaches, it will be found that

the patient will not repeat the numbers correctly until the examiner is slightly nearer than if the opposite method is adopted. However, the difference is not usually of great consequence, but one should accustom himself to a uniform method. There is also considerable variation in the different numbers, when spoken with equal loudness, some being heard at a greater distance than others. Nevertheless, the test is fairly accurate and remains the most useful one by which the degree of impairment of hearing may be determined.

FIG. 32.—Barany's noise apparatus.

Voice.—When the whisper cannot be heard near the ear the voice is used, the conversational or the shouted voice, according to the degree of impairment of hearing. When one ear is very deaf and the other but slightly so, it may be impossible to exclude hearing in the good ear with the finger. If, after testing a patient in this manner, the finger is inserted into the meatus of the ear under examination and it produces no change in his answers, it is fair to assume that he had been hearing the spoken words with the opposite ear. In such a patient a noise producer may be used. There are various forms of this apparatus, perhaps the best known is Barany's (Fig. 32). This winds like a clock and produces

a rattling noise which is conveyed to the ear by inserting the hard-rubber tip into the meatus, and produces so much noise that even the shouted voice cannot be heard on the side to which it is applied. When this is inserted into the good ear it is excluded and the test of the opposite ear becomes of value.

Further tests are made to determine whether the lesion causing the deafness is located in the sound-conducting or the sound-perceiving mechanism. The sound-conducting mechanism is that part of the auditory apparatus which transmits the sonorous vibrations to the labyrinthine fluid, and consists of the external auditory canal, the membrana tympani, the middle ear including the ossicular chain, and the oval and round windows; the sound-perceiving mechanism is that part which analyzes these vibrations and interprets them as sound and includes the acoustic labyrinth, the cochlear part of the eighth nerve and its central connections.

Fig. 33.—Dench fork for obtaining the lower tone limits.

Tone Limits.—The normal range of audition is from 16 double vibration (written D. V.) to upward of 30,000. The lower tone limit is determined by a large tuning-fork. For ordinary work the Dench fork answers very well (Fig. 33). This is a large fork with clamps on the prongs. With these extended as far as possible about 26 double vibrations per second are produced; with the ends of the clamps even with the prongs 32 D. V.; with the clamps removed 64 D. V. The clamps in any fork have a great influence in eliminating overtones, so when using a fork with them removed one should make sure that

overtones are not present, as the patient may be able to hear them when he could not perceive the fundamental note of the fork. If after the fork is set in vibration the base of the prongs is lightly touched with the finger, the overtones will be eliminated. These low-pitched forks cause a vibration of air against the patient's ear or drum membrane, and he may interpret this sensation as sound and say that he hears the fork, when he has not heard the fundamental note at all. Only after he has described the sound should it be concluded that he has heard it. By the Dench fork it may be ascertained that the lower tone limit is below 26 D. V., which indicates that so far as this limit is concerned there is no marked variation from normal. On the other hand, if the lower tone limit is above 32 or 64, it indicates disease of the sound-conducting mechanism. Marked impairment of hearing in a patient whose lower tone limit is below 26 D. V. is evidence of involvement of the sound-perceiving mechanism. For more accurate tests of the range of audition the Bezold-Edelman continuous tone series is advised. This consists of a set of ten tuning-forks with clamps which slide upon the prongs by which device a practically continuous tone series from 16 double vibrations per second to 1024 D. V. per second is obtained. For testing the tone perception above this point there are two closed pipes and an improved Galton whistle. It is possible with this set to determine the exact lower tone limit.

Upper Tone Limit.—The upper tone limit may usually be determined by the Galton whistle (Fig. 34). This instrument is so constructed that the pitch is altered by turning a screw and sounds are thus emitted which represent the upper part of the musical scale. If the opposite ear is closed as completely as possible, and the screw gradually turned at the same time by intermittent pressure on the bulb, a succession of high-pitched chirps are emitted. As soon as the point is reached in the descending scale at which the patient recognizes the sound as a musical note, the upper tone limit is determined. The

blowing, hissing sound produced incidentally by the instrument is to be ignored. The examiner ascertains that the patient has perceived the musical, high-pitched note by having him describe it, which he usually does, likening it to a whistle, chirp, or squeak. As the closed tube in the instrument is lengthened and the pitch thus lowered, the length of the tube is registered on a graduated scale. This number may be noted as upper tone limit 1, 2 or 3, etc., as the case may be; or if the Edelman instrument is used, it may be written in number of vibrations per second, this being ascertained from a corrected chart which accompanies the instrument. If it is desired to ascertain the number of vibrations in the ordinary instrument, it may be done, although roughly, by remembering

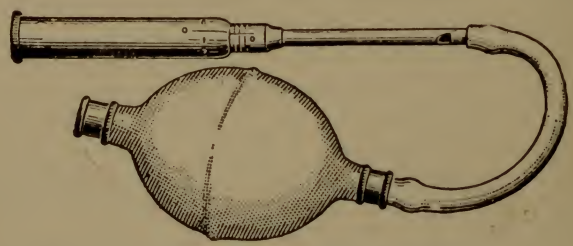

Fig. 34.—Galton whistle modified by Dench.

that according to the law of closed tubes, the tube length is one-fourth of the length of the sound wave.[1] For example: if when the patient perceived the note the tube length as shown by the graduated scale is one-fourth inch, the sound wave would be one inch, and as sound travels at the rate of (approximately) 1100 feet per second, that is, 13,200 inches, this would be the number of vibrations per second. Lowering of the upper tone limit indicates disease of the sound-perceiving mechanism.

Gaps and Islands.—At times the tone perception may be lost throughout the greater part of the musical scale, so that only notes pitched in a small part of the register

[1] Dench, 3d edition, p. 149.

are perceived. There may be one or more of these islands. Then, again, the patient may hear throughout the greater part of the musical scale, being deaf for notes pitched in a small segment only. There may be one or more of these tone gaps. Tone islands and tone gaps indicate disease of the sound-perceiving mechanism.

Bone Conduction.—In the normal ear bone conduction (written B. C.) is about half of air conduction (A. C.). There is considerable variation in the ratio of B. C. to A. C. with forks of different pitch. As a rule, the higher the pitch of the fork, the shorter is the duration of bone conduction as compared with air conduction and *vice versa*. The best fork for these relative tests of bone conduction is an unclamped one making 256 D. V. per second. If a good fork of this pitch is set in vibration it will be heard by the normal ear for about 40 seconds. If when it is struck the handle is placed on the mastoid process, it will be heard for twenty seconds; if then held to the ear, it will be heard for twenty more seconds. That is, B. C. = $\frac{1}{2}$ A. C. In diseases of the sound-conducting mechanism the bone conduction is increased both as compared with air conduction and with bone conduction in the normal ear. If in a normal person, after the fork is set in vibration, it is placed upon the vertex, the sound will be conducted to both ears with the same degree of intensity and will be heard about equally plain on the two sides. If, now, the finger is placed in one meatus, the sound immediately appears louder on that side. The finger in the ear acts very much as a lesion of the conducting mechanism. There are two explanations of this increase of bone conduction in middle- and external-ear diseases which produce deafness. The impairment of hearing is evidently due, in these diseases, to an obstruction to the entrance of sonorous vibrations which, therefore, cannot reach the labyrinthine fluid to set it in vibration unless they are of sufficient intensity to overcome the obstruction. If, therefore, the labyrinthine fluid is caused to vibrate by impressions conducted through

the bone, the organ of Corti with its central connections will be able to interpret these vibrations as sound. These intralabyrinthine vibrations naturally dissipate themselves by radiation in various directions but chiefly through the middle ear and external auditory canal, but, meeting with the same obstruction in these locations which causes the deafness, the vibrations are reflected back to the labyrinth and thus intensify the effect produced by the fork behind the ear or on the vertex and produce an increased bone conduction. Another explanation which has been offered is that the sound produced by bone conduction is intensified by the vibration of the column of air internal to the obstructing lesion. One can readily understand how this may take place when the cause of the deafness is occlusion of the meatus, as, for instance, a plug of cerumen, but this same phenomenon occurs when the lesion is at the foot-plate of the stapes on the very confines of the labyrinth and consequently there is no column of air between it and the labyrinthine fluid. So while some action cannot be denied to the vibration of the column of air it is probably of secondary importance in producing increased bone conduction. The increased bone conduction has given rise to three well-known tests: Rinné, Schwabach, and Weber.

Rinné Test.—The Rinné test is a comparison of the bone conduction with the air conduction in the same ear. The normal reaction is a positive Rinné; with a 256 D. V. fork B. C. = $\frac{1}{2}$ A. C. If the bone conduction increases and the air conduction diminishes, as it does in diseases of the sound-conducting mechanism, it may reach the point of reversal and B. C. will be greater than A. C., written B. C. > A. C. This is a negative Rinné. This usually occurs only when the impairment of hearing is marked. It may be expected when the forced whisper is heard at four feet or less. With less deafness than this the obstruction to the entrance of sonorous vibrations aërially conducted and the increase of bone conduction are not sufficient to reach the point of reversal; although

there may be a change in the normal ratio of bone conduction to air conduction. For example: B. C. = $\frac{2}{3}$ A. C. This is a positive Rinné but a diminished positive and indicates disease of the sound-conducting mechanism. In the above remarks it is supposed that a good fork giving 256 D. V. is used. In disease of the sound-perceiving mechanism the Rinné is positive, that is, B. C. $<$ A. C.

Schwabach Test.—In this test the bone conduction of the ear examined is compared with that of the normal ear, for instance, that of the examiner. The test has absolutely nothing to do with air condition. There are two ways of making this test, according to whether the patient's bone conduction is increased or diminished. The fork is struck and the handle placed upon the patient's mastoid process over the antrum; when he has ceased to hear it, it is transferred to the examiner's mastoid; when if heard, the bone conduction in the patient is less than normal, B. C. —. If the examiner, who is presumed to have normal bone conduction, fails to hear the fork, he may either have the same amount of bone conduction as the patient or less. To ascertain the condition the fork is again struck and if after the examiner ceases to hear it the patient does so, it is evident that the patient's bone conduction is increased, B. C. +. The amount of increase or diminution of the bone conduction may be taken in seconds, if desired. In order to ascertain the exact time at which the patient ceases hearing the fork he is directed to raise his hand when this occurs. He is also cautioned to be certain that he no longer hears the fork before doing this. The patient often seems to be uncertain on this point, either stating that he hears the fork when he does not or stating that he does not hear it, when if it is removed and then replaced he will hear it. This latter case may be due to fatigue of the auditory nerve in some instances but is far more often an evidence of inaccuracy on the part of the patient. If one ear is very deaf from disease of the sound-perceiving mechanism and the bone conduction in the other ear is good, the patient may hear the tuning-fork through the

head on the good side. So it is well, in order to avoid error, to ask the patient in which ear he hears the fork, which is placed upon the mastoid of the deafer ear. Increased bone conduction as obtained by the Schwabach test is an evidence of disease of the sound-conducting mechanism, and diminished bone conduction is evidence of disease of the sound-perceiving mechanism.

Weber Test.—The Weber test is a comparison of the bone conduction on the two sides. In order to have it successful there must be quite marked inequality of hearing in the two ears and the patient must be fairly intelligent. The fork is struck and placed upon the vertex or against the teeth. The patient will hear it best in the deafer ear if there is disease of the sound-conducting mechanism. It may be written Weber + R. or L., as the case may be, or B. C. R. > L. or B. C. R. < L., as the case may be. The statement of the patient is not always reliable in making this test, so if there is doubt of its accuracy it may be checked up with the Rinné and Schwabach tests.

Both the Rinné and Schwabach tests are more or less unsatisfactory in patients over fifty years of age. As age advances bone conduction becomes less. It may be very poor in patients as young as forty-five, then again some patients of sixty have very good bone conduction. But it is uncertain in patients over fifty and so the tests in which its duration is an element becomes unsatisfactory. If in making these tests the patient is found to react as in younger subjects, reliance may be placed upon them. If, however, his bone conduction is evidently reduced, no deduction from this fact can be made. In locating the lesion in the sound-conducting mechanism in these older patients, reliance must be placed upon the occurrence of better bone conduction in the deafer ear, and upon elevation of the lower tone limit; in locating the lesion in the sound-perceiving mechanism, reliance must be placed upon bone conduction being better in the good ear, lowering of the upper tone limit, the occurrence of tone islands or gaps.

Gellè Test.—This test is performed by setting the fork in motion and placing the handle on the skull. The air in the meatus is then condensed with the otoscope, or some similar apparatus. This forces the drum membrane and ossicular chain inward, leading to an increase in intralabyrinthine pressure. For this to take place it is supposed to be necessary for the stapes to be movable in the oval window. As the bulb is compressed, the fork should be perceived less plainly or not at all. If this occurs, the test is positive. A positive Gellè ought to indicate stapedial mobility. A negative test does not, however, indicate stapedial fixation, as other conditions may prevent the force exerted against the tympanic membrane from being conveyed to the labyrinthine fluid.

Bing's Experiment.—Another test not commonly in use is Bing's experiment. If the bone conduction in a normal patient is taken and when he ceases to hear the fork the finger is inserted into the meatus, it becomes audible again. In marked deafness from conductive-mechanism disease this may not occur, as the finger may not offer any added obstruction to the exit of the vibrations and produce no increase in bone conduction. If the fork is not again heard by bone conduction when the finger is inserted, it is evidence of a lesion in the sound-conducting mechanism. However, not much reliance can with safety be placed upon this test in making a diagnosis of the location of the lesion.

CHAPTER III.

DISEASES OF THE EXTERNAL EAR.

MALFORMATIONS.

Polyotia.—Malformations of congenital origin may be limited to the auricle or the external auditory canal also may be affected. In polyotia more than one ear exists on the same side. One may be perfectly formed while the other is rudimentary, or both may be rudimentary. In the former case the meatus is usually properly formed and the surgeon is consulted on account of the deformity caused by the presence of the supernumerary ear. This may exist in the form of an extra lobule, or it may not resemble any of the structures of the auricle, merely being in the form of a tag which may or may not contain cartilaginous tissue. These tags are usually located anterior to the auricle (Fig. 35). They may be removed by an elliptical incision for the cosmetic effect. When both of the auricles are rudimentary various deformities of the deeper structures are usually present which are discussed under Microtia.

Macrotia.—In macrotia there is an abnormal development of the auricle, either the pinna as a whole being too large or the increase in size is limited to certain parts of the ear, usually the lobule or the upper part of the auricle. Considerable variation in the size of the pinna is possible without the production of deformity providing the two ears are of the same size. If the auricles were symmetrical, although abnormally large, one would hesitate before operating upon them, as there is more or less danger of perichondritis in operations upon the auricle.

If the lobule is very large a wedge-shaped piece may be removed and the parts sutured or held in place with Michel's clamps. When the enlargement is in the upper part of the auricle a V-shaped piece may be removed and the margins of the wound brought together by two rows of sutures, one on the anterior, and one on the posterior surface of the auricle.

FIG. 35.—Polyotia. The auricle and canal normally developed. The tags represent a supernumerary auricle.

Microtia.—Microtia is a term applied to abnormally small ears. As usually seen, they are very much deformed. There may be a rudimentary lobule, tragus and the upper part of the helix, or the auricle may be so deformed that it is impossible to make out any parts corresponding to the normal organ. In subjects with this deformity there is usually occlusion or total absence of the external auditory canal. The Eustachian tube may be patent and the tympanum normal, or these parts may be more or less deformed. Malformations of the inner ear may accompany

microtia, but this is not the rule. The condition results in more or less profound impairment of hearing. If both sides are affected, the question of operation arises to gain, if possible, a useful degree of hearing. While the report of operations has not been generally favorable, still success has been attained in some instances. The operation should be undertaken if the patient's parents desire it after being informed as to the uncertainty of the result. It should not

Fig. 36.—Microtia (before operation). Dr. Page's patient (with permission).

be advised unless a trace of hearing is present and the bone conduction good, as if the labyrinth is simultaneously involved no good will be accomplished. The auricle is turned forward by a posterior incision. The mastoid antrum opened as widely as possible and the bone removed as far forward as would correspond to the anterior wall of the canal. The middle ear is thus opened. A good-sized meatus is formed in the skin and the wound lined

with Tiersch grafts. The cuts show the result of a case operated upon by Page[1] (Figs. 36 and 37).

Prominent Ears.—Occasionally the aurist is called upon to relieve the deformity caused by prominent ears. If seen in infancy the wearing of a tape cap may be advised.

Fig. 37.—The same ear as in Fig. 36 after operation. With permission.

This is so constructed that when in position the auricles are held in contact with the head by tapes passing over them. Continued wearing of this device results in the ears assuming a more normal position. The caps may be purchased in shops specializing in children's goods. In

[1] Tr. Am. Otolog. Soc., 1914.

adults the deformity may be due to a redundance of the cartilage or merely to a laxity of skin and contiguous tissue. In the former case in order to correct the deformity it is necessary to remove some of the cartilage or, as in an operation devised by Goldstein, to reduce the size of this structure by overlapping it. A curvilinear incision is made on the posterior surface of the auricle and the skin separated from the cartilage. This is now incised in a manner similar to that adopted in submucous resection of the septum, the incision merely passing through this structure and not the soft parts on the anterior surface of the auricle. These are now separated from the cartilage, the flaps of which are made to overlap and held in position by mattress sutures. The skin incision is then closed. Great care must be taken to avoid infection or perichondritis may result. If the soft tissues only are at fault an elliptical piece may be removed, of which part is taken from the posterior surface of the auricle and part from the tissues on the side of the head, or over the mastoid, and the incision brought together with deep and superficial sutures. Care must be taken to so locate this removal of tissue that the ear will assume a proper position when the flaps are brought together.

Fistula.—This condition occurs as a small pit usually in front of the auricle and is due to an incomplete closure of a branchial cleft. These fistulæ vary in depth from one-fourth of an inch to an inch and secrete a material not unlike sebaceous matter, which may be discharged externally or accumulate and become quite hard. At times they become inflamed, although usually they cause no discomfort. The inflammation will subside if the cavity is evacuated. For their radical cure they may be curetted, packed with gauze and allowed to heal from the bottom, but their total removal is advisable as resulting in less deformity. An elliptical incision is made and the walls of the fistula completely dissected out. The skin is brought together with clamps or sutures. Healing should result with an insignificant scar.

Bifid Lobule.—Bifid lobule is usually due to the tearing out of an ear-ring, but one is occasionally seen in which the patient denies all history of traumatism (Fig. 38). They may easily be remedied by freshening the separated margins and holding them together with sutures or Michel clamps.

FIG. 38.—Lobule in three parts. The patient denies traumatism. Her daughter has a bifid lobule and also denies traumatism.

CUTANEOUS DISEASES OF THE EXTERNAL EAR.

The integument of the auricle and canal is subject to the various cutaneous diseases. It may be affected as part of a general cutaneous involvement when the patient will probably be seen by the dermatologist; or the ear alone may be affected when the otologist is usually consulted.

Intertrigo.—This is usually a disease of infants and younger children and occurs in the fold behind the ear. It is caused by the contact of the opposing surfaces, either due to the natural position of the parts or brought about by the wearing of caps which press the auricle against the mastoid region. The parts become red and moist from the

inability of evaporation of the natural secretion to take place, the epithelium becomes macerated, exfoliates and decomposition occurs. Another form is caused by wearing bandages which are necessary after mastoid operations. These act by preventing the evaporation of the natural moisture which is augmented by the secretion from the mastoid wound. In this manner is produced maceration of the epithelium, irritation of the skin and exudation of a discharge, which decomposing becomes more or less odoriferous. Either of these forms may result in acute eczema, the latter being usually associated with it when well developed. Lack of cleanliness is also without doubt a frequent cause.

Symptoms.—The symptoms are burning and pruritus. The child is continually trying to scratch the parts, and if there is a bandage he is trying to insert his finger beneath it and usually succeeds in infecting the mastoid wound if the bandage is not properly applied.

Treatment.—The treatment consists in cleanliness and keeping the parts dry and separated. In the first form the parts are cleansed with soap and water not oftener than once a day. Subnitrate of bismuth or stearate of zinc may be dusted on every four hours or as often as the parts show a tendency to moisture. The wearing of caps binding the ears is discontinued. If the disease proves stubborn, oxide of zinc ointment may be used, and if necessary the parts may be separated by a small piece of lint upon which the ointment has been spread. When this condition occurs under mastoid dressings, treatment is often unsuccessful until the bandage can be left off. When changing the dressings the parts are to be thoroughly cleansed with pledgets of lint, and afterward dusted with stearate of zinc, or oxide of zinc ointment may be spread over the parts with a glass spatula or upon gauze which is applied to the involved surface. The bandage should be drawn tight over the cheek and neck to prevent, if possible, the child from inserting his fingers and producing further infection.

Eczema.—Eczema may be acute or chronic. The acute form is more often seen by the aurist, although occasionally it will have become chronic before it comes under observation. The chronic form is more apt to be but part of a general eczema of the face or scalp. Acute eczema insofar as it has a local cause, is due to the irritation from an aural discharge or from applications made by the patient, in the form of drops, liniments or ointments, for the relief of aural symptoms. It begins with the formation of vesicles, which rupture and exude a tenacious serum which dries into crusts or scales. If the disease progresses to a cure, these drop off and leave a red surface which gradually returns to normal. If the disease becomes subacute or chronic, as these scales and crusts come away, a weeping surface is left and new crusts form; the deeper layers of the skin become infiltrated and considerable thickening of the auricle or canal results, in the latter situation leading at times to a marked narrowing of the canal lumen.

Symptoms.—The symptoms are a smarting sensation and pruritus. If the patient gives way to the desire to scratch the parts, the crusts may be torn away, resulting in bleeding and the formation of new crusts mixed with dried blood. Scratching the canal with ear spoons, matches, heads of pins, or the finger nail not infrequently results in infection and the production of a circumscribed otitis externa. When irritation from a middle-ear discharge causes the disease, the canal is moist and the formation of crusts does not take place, and there may be an area adjacent to the meatus in the same condition which appears red and denuded of epithelium. At the margin of the denuded area appear the crusts and scales characteristic of the disease.

Treatment.—In treating this disease in patients who have no middle-ear discharge, the first endeavor is to ascertain whether the process is in the acute stage and demands sedative and emollient treatment, or whether it is more chronic and demands stimulation. In the

acute stage before the formation of crusts oxide of zinc ointment answers very well. This is applied twice a day. If the disease extends into the canal and it seems necessary to apply the ointment to this region, the patient may make a small applicator by winding cotton on a tooth-pick, with which the ointment is lightly applied to the canal walls. It is usually a bad plan, however, to apply oleaginous preparations to the meatus, and they should be avoided if possible. When crusts have formed in acute processes, they may be removed with soap and water or with olive oil, after which oxide of zinc ointment, to which has been added oil of cade 5 to 10 drops to the ounce, may be applied two or three times daily. When the disease has become chronic or threatens to do so, the amount of oil of cade is increased as indicated from 15 to 30 or even 60 minims to the ounce, according to the amount of stimulation desired, and the crusts may be removed with green soap.

For eczemas resulting from middle-ear discharge, as the condition is frequently caused by its improper removal, cleanliness is essential. The secretion is to be removed every four hours or even oftener by syringing the ear with a saturated solution of boracic acid or bichloride, 1 to 5000, after which the canal is to be dried and stearate of zinc or boracic acid applied by insufflation. The external parts are covered with oxide of zinc ointment to which the proper amount of oil of cade has been added. This may also be applied to the canal walls as advised above if the process proves stubborn. It will, however, but seldom be necessary. Nitrate of silver, 2 to 6 per cent. solution, may be applied to the canal once a day when other means fail. It should not be applied to the auricle on account of the discoloration which it causes. In chronic eczema, painting the auricle with cantharidal collodion often works well when other means fail. It should not be used oftener than every two to four days, according to the effect produced. The list of remedies which have been advocated is a long one. The

treatment outlined above will, however, usually be successful. The general health should be attended to, and any measures adopted which seem necessary. Cure may often be hastened by the administration of arsenic, iodide of iron, and cod-liver oil.

Dermatitis.—This is an inflammation of the skin of the auricle due to irritants of various kinds. The most common causes are: the application of heat by poultices, hot-water bottles or the direct rays of the sun; irritating liniments and ointments; freezing the ear by exposure. Any irritant, chemical or mechanical, is capable of producing this condition. Why in a given instance simple dermatitis rather than eczema is produced is at times difficult to understand. Some underlying systemic or local cause is probably necessary for the production of the latter condition. Varying with the degree of the irritation, simple redness of the skin, vesicles or inflammation of the deeper dermal layers may result. In severe burns and frost-bite destruction of the cartilage may accompany the dermatitis.

Symptoms.—The symptoms vary from a burning, itching sensation to actual pain. If the cartilage is exposed, infection may occur, resulting in perichondritis. Upon inspection, the parts may be simply reddened or vesicles may be present, or the whole auricle may appear thickened and tender. If sloughing has occurred, the cartilage may be exposed.

Treatment.—The treatment varies with the severity of the inflammation. In the milder forms some emollient ointment or cold cream is very efficacious. In the severer forms, application of compresses saturated in a 4 to 8 per cent. solution of aluminum acetate, or acetate of lead, 4 grs. to the ounce, will often prove efficacious. If itching is severe, a solution of carbolic acid, 1 part in 100 of water, may be used. It is a matter of common knowledge that a frost-bitten ear should be "thawed out" with snow to prevent reaction. In the severer cases where there is destruction of skin with exposure of car-

tilage, the sloughs should be removed and the cartilage if necrotic dissected out, and the wound otherwise treated on general principles.

Erysipelas.—Erysipelas is an inflammation of the skin, caused by infection. As usually seen by the aural surgeon, it develops subsequent to some operation on the mastoid. It is due to infection with a streptococcus, but does not seem to occur with much more frequency after operations in which a streptococcus is the prevailing germ than in those in which this type of organism is absent.

Symptoms.—The disease is usually ushered in by a chill which is followed by fever and it may be a day or so before the cutaneous lesions are sufficiently developed to enable one to make a diagnosis. In the meantime much anxiety is caused, as these same symptoms may occur with certain intracranial involvements following operations on the mastoid. There is usually pain and tenderness in the involved parts. Upon inspection there is a dull red, brawny infiltration of the skin which shows a well-marked line of demarcation. The auricle may be very much thickened and when the disease is fully developed is covered with vesicles. Deep involvement of the middle ear or mastoid rarely occurs. It is not uncommon to see the grafts take well after a radical operation notwithstanding this disease.

Diagnosis.—The diagnosis is made from simple dermatitis by the temperature and the presence of the line of demarcation. When this has formed it is characteristic. Occasionally the reaction caused by iodin which has been employed previous to operation closely resembles erysipelas. It will be found, however, that this does not extend from day to day, nor does it produce fever. The disease necessitates isolation. When they return from the isolation ward it is usually found that the wound has progressed satisfactorily.

Prognosis.—The prognosis is usually good. Most of the patients get well, although the disease is by no means free from danger.

Treatment.—The treatment consists in the application of a solution of acetate of lead, or aluminum acetate. The general condition of the patient should be attended to. Tr. chloride of iron in full doses still remains as good as any internal treatment. The administration of the leukocyte extract (His) has seemed to aid to a cure in some instances. As the patients usually get well it might naturally be expected that many forms of treatment would claim the credit—and this is the case.

HERPES OF THE EXTERNAL EAR.

Herpetic eruptions of the external ear, as compared with herpes zoster in other regions, is of uncommon occurrence. Herpes zoster may form in the area of distribution of any of the cutaneous nerves which supply the auricle. The eruption may be located on the neck and head as well as on the posterior surface of the auricle in cervico-occipital involvement or on some part of the face and temporal region as well as on the anterior surface of the auricle in facial herpes.

Herpes Zoster Oticus.—In one form distinct from these types, known as herpes zoster oticus, the lesions are more or less limited to the anterior surface of the auricle and auditory canal. It is usually accompanied by facial paralysis. In a patient of the author's, who had this type of herpes, the eruption was practically limited to the region of the concha, only a few vesicles appearing upon the antihelix and antitragus, the meatus and membrana tympani being free. Ramsey Hunt[1] has brought this type to the notice of American otologists. He believes that the eruption occurs in the zoster zone of the geniculate ganglion and divides herpes zoster oticus into four groups: (1) herpes oticus; (2) herpes oticus with facial paraylsis; (3) herpes oticus with facial paralysis and hypo-acousis; (4) herpes oticus with facial paralysis and Ménière's

[1] Journal of Nervous and Mental Disease, February, 1911; American Jour. Med. Sc., August, 1908.

symptom-complex. It is assumed that trophic and sensory nerves accompany the facial and are distributed to the skin of the auricle and canal. The geniculate ganglion is considered as analogous to the ganglia on the posterior roots of the spinal nerves, the pars intermedia of Wrisberg being the sensory root. This hypothesis accounts for the four varieties given above. Involvement of the ganglion, the motor fibers of the facial not being implicated, produces neuralgia and the herpetic eruption. If the motor fibers are also involved, facial paralysis is added to these symptoms. In the third form, in which there is hypo-acousis or diminished hearing, this symptom is due to involvement of the cochlear branch of the eighth or perhaps to the peripheral ganglia (spiral ganglion) which exists on this nerve. In the fourth group the vestibular branch of the eighth would be involved, in addition to the cochlear, accounting for the deafness, tinnitus, vertigo, vomiting and staggering gait, this being Ménière's symptom-complex.

Symptoms.—Herpes begins with malaise and fever, usually of mild degree. Pain more or less severe follows. In a varying time, from one to several days, the parts become reddened, which is soon followed by the appearance of vesicles. These dry into scales and fall off in about a week, leaving a reddened base, which gradually fades. If facial paralysis occurs, it usually develops about the time of the appearance of the vesicles. The pain may subside when the vesicles appear or continue after they have healed. In some patients there is swelling of the auricle and external auditory canal and the eruption may occur on the membrana tympani and canal walls.

Diagnosis.—The diagnosis offers no difficulty. The occurrence of pain with the characteristic eruption can hardly be mistaken for any other disease.

Prognosis.—The prognosis is usually good. The eruption heals, the pain usually passes away and the facial paralysis, although in some patients quite persistent, usually gets well.

Treatment.—The treatment is antineuralgic. Phenacetin, grs. v, every four hours, or aspirin, grs. x, four times a day, will be found useful. The ordinary migraine tablets, one every two hours, frequently ameliorate the pain. These contain acetanilid, 2 grs., monobromate of camphor and citrated caffeine ½ grain of each. Electricity is at times of value to relieve the pain. It should be used both for this object and to maintain the nutrition of the facial muscles, in those patients in whom palsy is present.

OTITIS EXTERNA HEMORRHAGICA.

Closely allied to herpes in its symptoms and clinical course is the condition known as otitis externa hemorrhagica. The lesion characteristic of this disease is produced by a circumscribed effusion of blood or bloody serum beneath the epithelium of the external auditory canal. The favorite location of these blebs is on the floor of the canal near the drum membrane, although they may occur on any part of the canal wall or membrana tympani. They vary greatly in size and number in different patients. They are of a dark blue color and do not seem to rest upon an inflamed base. One is frequently impressed with their resemblance to large herpetic vesicles containing blood in place of serum.

Symptoms.—The most prominent symptom is pain. This is usually severe and sufficient to keep the patient awake at night. If the lesions are on the drum membrane, there is usually some deafness and tinnitus, but these symptoms do not usually cause much complaint on the part of the patient. Occasionally a patient comes on account of the bloody discharge which is caused by the rupture of the blebs. In patients seen by the writer the middle ear has usually been free from involvement, although Politzer states that he has often observed the disease accompanying otitis media of influenzal origin.

Diagnosis.—The diagnosis is usually easily made. There is really no disease with which it can be confounded. The severe pain associated with hemorrhagic blebs is characteristic of this disease alone. It is not always easy to form an opinion as to the condition of the middle ear, when the lesions are situated on the drum membrane. Inflation may demonstrate that the tympanum is free from fluid, thus giving evidence that it is not involved.

Prognosis.—The prognosis is good. The patients usually recover in from a few days to a week or so.

Treatment.—The treatment is antineuralgic. Phenacetin, aspirin, or the neuralgic tablets may be given in the same manner as in herpes. The local treatment consists in opening the blebs, wiping the blood and serum away, and dusting the parts with boracic acid, or the blebs may be allowed to take care of themselves. In patients with the lesions on the drum membrane, especially if accompanied with severe pain, if there is any doubt as to there being middle-ear involvement, a free myringotomy should be done after which the patient is treated the same as in otitis media.

OTALGIA.

Neuralgia affecting the region of the ear is termed otalgia. It may be a part of a general trigeminal or cervico-occipital neuralgia or the pain may be localized in the ear. When the auriculotemporal is involved, the pain is referred to the anterior surface of the auricle and pre-auricular region, while if the auricularis magnus and small occipital are affected the posterior surface of the auricle and mastoid region are the seat of the pain. There may be general hyperesthesia over the area supplied by the nerve, or tenderness may be limited to one or more points. The otalgia may be located in the deeper parts of the meatus or the tympanum, in which event the tympanic plexus is supposed to be involved.

Etiology.—The predisposing causes of otalgia are anemia, malaria, hysteria and neurasthenia, but it is by

no means infrequent in patients in whom none of these causes are operative. The most frequent exciting cause is some abnormal condition of the teeth, such as caries, eruption of molar teeth, bridge-work, or an impacted tooth not growing properly. Inflammation in and around the tonsil often produces severe pain in the ear, as does also involvement of the temporomaxillary articulation. Scars resulting from mastoid operations are occasionally the cause of severe neuralgic pain.

Symptoms.—Some otalgias are of short duration. These are naturally those in which the cause can be found and removed. Others are chronic, recurring at intervals through months or years. It is not uncommon to see patients who complain that they have suffered from pain in the ear for months. The only important symptom is pain. This is usually quite intense during the height of the paroxysm, but is not as continuous as in inflammatory affections of the middle ear and mastoid. It is, however, usually sufficiently severe to keep the patient awake at night.

Diagnosis.—The diagnosis is made upon the presence of pain and the absence of changes in the drum membrane and meatus. When there is hyperesthesia over the mastoid region the tenderness may quite closely resemble that found in mastoid disease. The absence of signs of middle-ear involvement as well as the fact that the hyperesthesia of otalgia is apt to extend along the well-known course of a nerve and outside of the mastoid area enable one to make a diagnosis. If the patient has instilled drops into the ear and these have produced changes in the membrana tympani, difficulty will arise. The aid of the *x*-rays may be invoked when doubt is experienced.

Prognosis.—When the cause can be found and relieved the prognosis is good. This is possible in the majority of patients who come under the care of the otologist. Of the minority some will be cured, while in others there may be recurring attacks of pain for some time.

Treatment.—In treating these patients search is made for the cause. The teeth demand the first attention. If they are not evidently carious or an erupting tooth cannot be found an *x*-ray is taken. This may reveal a carious area, unsuspected before, or that the teeth are impacted and growing improperly. Inflammatory affections of the tonsils are to be treated and peritonsillar abscesses opened. When the temporomaxillary articulation is involved, counterirritation and antirheumatic remedies may be used. When the cause cannot be found or to relieve the pain pending its removal antineuralgic remedies may be given, phenacetin, aspirin or the migraine tablets as directed under Herpes will be found useful. In the malarial patients, quinine and arsenic; in the anemic ones, tonics are to be administered.

HEMATOMA AURIS.

Hematoma auris is characterized by the effusion of blood or bloody serum between the perichondrium and the auricular cartilage.

Etiology.—It is common in the insane and in them seems to occur spontaneously. Just how much traumatism may have to do with causing it in these patients is undecided. That the mental state may have an effect seems probable in view of the experiments of Brown-Sequard, who found othematoma in animals in which the restiform body had been severed. As a result of traumatism it not infrequently occurs in the sane. Especially predisposed on account of their occupation are prize-fighters and acrobats. It seems probable that some degenerative change in the bloodvessels or cartilage is frequently a predisposing cause, as at times a severe trauma does not result in the production of an hematoma, while at others even a slight pull upon the ear is sufficient to cause one. These changes in prize-fighters and acrobats may be brought about by repeated traumatism.

The disease is more common on the left side, both in the sane and the insane, which is an argument in favor of its traumatic origin.

Symptoms.—Othematoma appears in the form of a tumor, either bluish or of the color of the normal skin, usually situated on the anterior surface of the auricle (Fig. 39). In the beginning these enlargements feel doughy and are not tender on pressure. At this stage

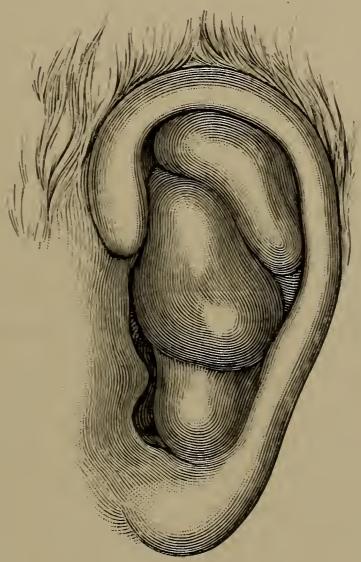

FIG. 39.—Othematoma. (Posey and Wright.)

the patients rarely complain of pain but come for the relief of the deformity caused by the tumor. Later there may be pain especially if infection occurs.

Diagnosis.—The only condition resembling this is perichondritis. The diagnosis is made from the history of traumatism, the presence of a well-defined tumor and absence of inflammation in hematoma. If transilluminated the hematoma will appear opaque while perichondritis is more or less translucent (Politzer). If

infection occurs the process becomes one of perichondritis and should be treated as such.

Treatment.—Spontaneous cure may result usually with more or less deformity. In the forming stage light pressure with the application of cold will limit the size of the effusion. After the tumor is well formed, aseptic evacuation, either by aspiration or if this is unsuccessful by a small incision followed by the application of an aluminum acetate dressing firmly applied, is usually followed by good results. If the swelling becomes painful and infection occurs or seems imminent a free incision should be made and the wound packed with gauze. At times even after the most skilful treatment deformity results from crumpling of the cartilage, and this whether or not perichondritis has developed.

PERICHONDRITIS OF THE AURICLE.

Etiology.—Common causes are furuncles, infection of the meatal incision in the radical operation and hematoma of the auricle; although any injury of the auricle which becomes infected may produce the disease. The staphylococcus and pyocyaneus are the organisms which are usually responsible for perichondritis. In a patient of the author's the condition was due to scarlatinal infection following the radical operation.

Pathology.—In this disease there is an inflammation of the perichondrium covering the cartilaginous framework of the auricle. As the lobule contains no cartilage, this is never involved. The region of concha adjacent to the meatus is frequently the part affected, mainly because the disease is more often due to processes within the meatus than to other causes, although any part of the auricle may be involved except the lobule. The inflammation of the perichondrium results in an effusion between it and the cartilage. This effusion is serous, colloidal, not unlike synovial fluid in consistence, or purulent, and as it forms the perichondrium is stripped

from the cartilage. This results in necrosis of the latter which thus becomes a foreign body.

Symptoms.—The symptoms are pain and a slight rise of temperature. The pain is not usually severe and the elevation of temperature is not more than two or three degrees. Upon inspecting the parts the auricle will be found thickened over the involved area, slightly red in color, tender on pressure with elevation of the local temperature. If effusion has taken place there will also be fluctuation. In the serous form, in which the effusion is usually limited, the reaction is less and the parts have a velvety feel. As the disease progresses and the effusion becomes greater and of a colloidal or purulent nature, a well-defined tumor is formed, which if neglected may rupture, resulting in the formation of fistulæ.

Diagnosis.—The diagnosis is to be made from hematoma which it resembles when a well-marked tumor has formed, by the inflammation which is present in perichondritis. The serous form may be mistaken for dermatitis, but the velvety sensation communicated to the fingers and the deeper location of the swelling in perichondritis usually enable one to differentiate them.

Prognosis.—After the process subsides even with the most skilful treatment some deformity of the auricle usually remains, which in patients who have had no treatment is often very marked (Figs. 40 and 41).

Treatment.—In the very early stages moist dressings of aluminum acetate may be applied. In the diffuse serous form this often affects a cure. If the effusion is localized and a tumor begins to form or signs of inflammation become well marked, a free incision should be made, the colloidal or purulent material evacuated and the necrotic cartilage removed with the curette. If in spite of this treatment the process extends so that more cartilage becomes involved, the incision should be enlarged and the necrosed cartilage dissected out. This is removed until healthy cartilage is encountered. This operation will result in minimizing the deformity, if it can be

accomplished without weakening the outer part of the auricular framework. If a substantial rim of sound

Fig. 40.—Patient showing the deformity following perichondritis. Note that the lobule is not involved.

Fig. 41.—The opposite ear of the patient shown in Fig. 40.

cartilage can be left, the operation is advisable; if not, little will be gained by it, and it is well to treat the process by free incisions and moist dressings.

IMPACTED CERUMEN.

Causation.—The ceruminous glands are situated in and beneath the skin in the outer or cartilaginous part of the external auditory canal. They are modified sebaceous glands and secrete a yellowish, oily material—the cerumen. It is bitter in taste and is supposed to be designed by nature to discourage the entrance of insects, etc. Normally it lies in a thin layer covering a very limited area at the entrance to the meatus and as more is secreted moves outward and is removed either in washing the ear or by the finger. At times, however, it accumulates and fills the meatus to a greater or less extent. This is due to increased secretion of cerumen of altered composition or to the shape of the canal. The movements of the inferior maxilla have an effect to stimulate the outward flow of the secretion but in some instances, in which cerumen is retained, the effect of the action of the condyle of the mandible upon the meatus seems to be directly the opposite, probably on account of the shape of the canal. As the secretion accumulates the mass is forced deeper into the canal either by the fresh secretion or by the patient's finger in his efforts to clear the ear until it may approach or even impinge upon the tympanic membrane.

Pathology.—It is a very common condition. In some patients after being once removed it does not accumulate again. Others seem to have a recurrence at quite regular intervals, showing that nature is unable to take care of it. The mass when removed consists of cerumen, more or less epithelial débris, sometimes various fungi. A foreign body, such as a piece of cotton, may be found in the center of a ceruminous plug. The mass at times retains the shape of the canal and is covered with the epithelial lining of the meatus which has desquamated and adhered to the cerumen. Masses of cerumen lying on the walls of the canal are very frequently seen while making aural examinations. They may interfere with a view of the

deeper parts, and are easily removed with the dull ring curette. Usually it is not until the mass totally occludes the canal that symptoms are produced. This is very apt to occur after introduction of water which causes swelling or moving the plug or the same result is brought about by efforts on the part of the patient to clean the ear.

Symptoms.—The usual symptoms are a feeling of fulness or stuffiness in the ear, a moderate degree of impaired hearing and tinnitus. The deafness will be much worse if the ceruminous plug has been forced into contact with the drum membrane, and in this event there may also be vertigo. The impairment of hearing will be found to be due to involvement of the sound-conducting mechanism and should be taken before the cerumen is removed and again after. In this way should there be other causes for the deafness an estimate may be formed of how much of the impairment of hearing was due to the cerumen. Cough may be caused by impacted cerumen. This is attributed to reflex action through the pneumogastric nerve brought about by irritation of its auricular branch (Arnold's nerve) which supplies the floor of the meatus. In all coughs not otherwise accounted for the ear should be examined. The color of the mass as seen on otoscopic examination is usually reddish brown, but may be white and glistening from being covered with epithelial scales or cholesterin crystals. The impression conveyed to one is that the mass is much nearer than the membrana tympani could possibly be. If there is any doubt, it can be dispelled by the use of the probe which will show that its consistence is semisolid.

Diagnosis.—The only condition apt to cause difficulty in diagnosis is a suppurative middle-ear process in which the secretion is so scanty that it dries in the meatus into a hard plug. Frequently, when the probe encounters these masses of dried pus they prove very hard. The diagnosis is not important, however, as the indications for treatment are the same in either event: namely, the removal of the mass.

Prognosis.—One must not be too sure of relieving his patient of all symptoms by the removal of the impacted cerumen, although he usually succeeds in doing so. The fact that these masses often occur in patients who have chronic catarrhal otitis media should be borne in mind, and also the possibility that they may consist of dried pus or cholesteatomatous masses from a chronic suppurative middle-ear process.

Treatment.—The best means to accomplish the removal of impacted cerumen is by the syringe, aided by the use of the ring curette, if necessary. Some aurists advocate the use of the curette alone. This, in many instances, will require great skill and be more disagreeable to the patient. There is practically no danger in syringing if properly performed. A warm antiseptic solution (bichloride 1 to 5000 or saturated solution of boracic acid) is used and the mass removed at one sitting. The preliminary use of drops to soften the cerumen is not advised. The patient is entitled to the immediate and complete removal of the offending mass as soon as it is discovered. In syringing the ear for the removal of cerumen the object to be attained is to force some of the solution between the mass and the canal walls so that, accumulating internal to the plug, it will force the cerumen outward. To accomplish this the auricle is drawn upward and backward and the stream directed against the canal walls near the cerumen. Considerable force may be used in this manner without danger. The current is directed alternately against the different walls of the canal, with the object of finding some location where it may pass between the mass and the wall. A good-sized syringe is used, holding two or three ounces (Fig. 42). Sometimes great aid is afforded by illuminating the parts with reflected light. Seeing the canal enables one to give the proper direction to the stream. If some headway is not made after reasonable efforts, the curette may be inserted between the superior or posterior wall and the mass and a part of the cerumen removed, or if possible a tunnel formed through

which the solution may be forced. Then by renewing the syringing and directing the stream at this point, the plug may be brought away. If still unsuccessful, more of the cerumen may be removed with the curette, and the syringing renewed. With patience, by this method one will succeed. After the cerumen is removed the canal is dried with the cotton applicator to absorb the solution remaining in contact with the drum membrane and a careful inspection of this structure and of the canal walls made. Furuncles are not of infrequent occurrence after the removal of cerumen. So if any abraded surfaces or points are found, they should be wiped with a solution of bichloride in alcohol (bichloride, gr. $\frac{1}{4}$, alcohol, 50 per cent., ℥j). This will also prevent the growth of

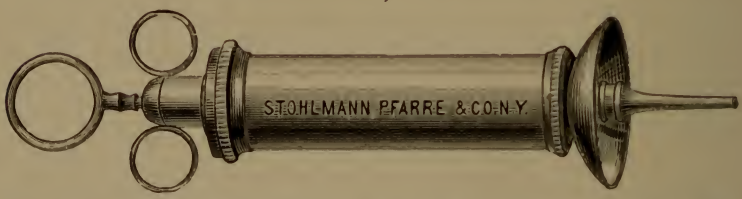

Fig. 42.—Large aural syringe for removing cerumen. Capacity three ounces.

fungi which at times form a considerable part of the ceruminous plug, the spores of which may still be adhering to the canal walls. If the weather is severe, a cotton wad may be worn in the meatus for twenty-four hours.

FOREIGN BODIES IN THE EXTERNAL AUDITORY CANAL.

Various kinds of foreign bodies find their way into the external auditory canal. Cotton, beads, buttons, various kinds of seeds, insects or their larvæ, pieces of pencil, small pebbles, are among the commoner ones. Some are left in the ear by mistake, but by far the larger number are introduced by children, who seem to have a special

disposition to get into trouble in this way. Otological literature is full of unique experiences with foreign bodies and the special means adopted for their extraction. They usually cause no trouble until unskilful attempts at their removal have been made. Exceptions are bodies that have been violently introduced, living bodies and seeds, as peas or beans which swell and cause pressure and occlusion of the meatus. It is not designed to minimize the seriousness of foreign bodies, for many of them are capable of causing great annoyance or even danger.

Symptoms.—If the foreign body completely fills the canal, or if this is closed by swelling, there will be more or less deafness and perhaps also tinnitus with a feeling of fulness or stuffiness in the ears. If the body is smaller and the parts have not been injured, it may produce no symptoms but become imbedded in the cerumen and remain for years unknown to the patient. On the other hand, such bodies may cause coughing and are supposed to have been the cause of epilepsy and various nervous disorders. If the canal wall has been injured, there will be swelling of the parts, with pain and a watery or bloody discharge. Rupture of the membrana tympani may be followed by otitis media with a purulent discharge. Bodies which enter the ear with force, such as bullets or pieces of metal, may penetrate the labyrinth and cause labyrinthitis and meningitis. In rare instances the body has been known to enter the tympanum through the Eustachian tube.

Diagnosis.—The diagnosis is usually made from the history and by finding the body by reflected light. Subjects offering difficulty are those in which efforts at extraction have injured the canal walls, producing an otitis externa with narrowing of the lumen of the meatus so that the body, already forced quite deeply, is with difficulty made out through the narrow canal surrounded, as it is, by blood and secretions. Before attempting to remove a foreign body its presence should first be determined. This advice is justified by the fact that the

instances are not infrequent in which attempts at removal have been persevered in, until damage has been done, the operator never having seen the body but relying upon the statement by the patient that "it must be in the ear."

Treatment.—Ordinarily the removal of the body if seen at once is not difficult. It is still near the entrance of the meatus, and can be rolled out with the dull ring curette or a small hook. This is not applicable if the body is sufficiently large to occlude the canal, as the curette cannot be passed between it and the canal wall. If it is oval in form its position may be changed so that the curette or hook may be inserted. If there is no moisture, some adhesive material may be used. A camel-hair pencil dipped in glue and applied to the surface of the body soon dries and hardens, so that it may be removed by traction. A cotton pledget, a piece of paper, a long flat object or one presenting a small process, may with safety be extracted with the forceps. To attempt to use this instrument upon a smooth oval or round object only results in the forceps slipping, thus forcing the body deeper into the canal, and if it passes the isthmus its removal becomes much more difficult. Advantage should be taken of any holes, such as there are in beads, buttons, etc., which will enable the operator to insert a hook, thereby facilitating extraction. In children an anesthetic may be necessary, at times even before they will permit an examination. This should be given, as nothing of value can be accomplished with a struggling child. Sometimes by syringing, the body can be removed, or its position so changed that extraction becomes possible. However, syringing as a matter of routine is not advised, although in the hands of those not possessing skill in aural work it is safer than any other procedure, and while it often succeeds, if it fails, no harm is done. Seeds such as peas or beans that have swollen and become soft may sometimes be extracted by first removing the contents of the capsule, when this is easily removed with forceps. Destroying the vitality of a living body with chloroform or a strong solution of bichloride of mercury

often facilitates its removal. A variation in the plan of procedure is often necessary to adapt it to special conditions. One finds use for all of his ingenuity in overcoming mechanical obstacles.

Posterior Incision.—When all other means fail, it may become necessary to remove the body through a posterior incision. This is made behind and close to the auricle which is drawn forward and the canal exposed. The membranous canal is separated from the osseous meatus posteriorly and above, and incised near the location of the body. This may give sufficient room to extract the body, although it will usually be found necessary to remove part of the posterior and superior bony canal walls with the gouge, before one is able to effect its removal. This operation affords a direct approach and a more roomy field. It is, however, a procedure of last resort, but it is much better to adopt it than to persist in useless attempts by the other methods. It is also available when bullets or pieces of metal have become imbedded in the tympanum or labyrinth, although, when this is the case, a much more extensive removal of the bone becomes necessary. After the operation is completed, the auricle is replaced and sutured posteriorly and the canal packed with gauze to prevent, if possible, subsequent contraction.

After-treatment.—After the removal of a foreign body by the other methods, the treatment varies according to the amount of injury done by the body or during its extraction. If the membrana tympani has been ruptured and the tympanum invaded, the treatment is the same as in otitis media; if the walls of the canal have been injured, the same as in otitis externa; while the simple cases require no treatment.

OTITIS EXTERNA DIFFUSA.

Definition.—Diffuse otitis externa is an inflammation of the meatus which, unlike furuncle, is not limited to any circumscribed area or areas. Two types are met

with. In one the inflammation involves the superficial layers of the skin, while in the other the deeper layers and the contiguous tissues are affected.

Superficial Form.—In the superficial form, which may be called desquamative otitis externa, the epithelium is cast off, frequently leaving the canal wall red and discharging a watery material, although at times when the epithelial mass is removed the base is found dry. It is due to irritation from middle-ear discharge, instillation of solutions or mechanical injury. At times it is caused by cerumen. One form of desquamation occurs in canals in which the cerumen is absent and is fairly attributable to the absence of this secretion.

Symptoms.—The symptoms are pruritus and in some patients a serous discharge. The desire to scratch the ear seems irresistible and its gratification only results in aggravating the process, and at times in the production of a circumscribed otitis externa. Pain is rarely complained of, although some soreness upon movement of the auricle is not uncommonly present. Inspection shows the exfoliating epidermis either upon a dry base or lying upon a surface exuding moisture. When due to middle-ear discharge, the epidermal masses may be absent, the denuded canal walls appearing red and inflamed.

Treatment.—The indications for treatment are to keep the parts dry and clean. The desquamating mass is to be removed and boracic acid or some other powder applied. In those processes caused by a middle-ear discharge, the parts are to be carefully dried after syringing and boracic acid insufflated. The mere cleansing of the parts in syringing has a beneficial effect. The pus should not be allowed to accumulate in the meatus and irritate the skin. In stubborn cases a saturated solution of boracic acid in 50 per cent. alcohol may prove efficacious.

Deep Form.—The deep form of otitis externa may not only involve the skin but the tissues adjacent to it. It

is caused by injuries of the canal walls, by infection, either from middle-ear discharge or introduced from without; or by chemical or mechanical irritants. Lowered resistance is undoubtedly a predisposing cause. Judging from a study of the foreign literature, it would seem that the severe varieties of this type of otitis externa are much more common in Europe than in America.

Pathology.—The process consists of inflammation and thickening of the canal walls, which may break down in places and cause abscesses and ulcers, from which granulations may spring. As usually seen, however, the process is in the stage of diffuse thickening of the meatus.

Symptoms.—The patient complains of pain which may be very intense. There is tenderness upon moving the auricle or pressure which moves the cartilaginous canal. There may be pain during mastication. There may be fever, but the rise of temperature is not over a degree or two, except in the very severe types. If occlusion of the canal occurs either as a result of swelling of its walls or from accumulation of secretion, there will be deafness, perhaps also tinnitus.

Diagnosis.—The diagnosis is made from furuncle by the fact that the thickening is diffuse, the swelling and tenderness not being localized as in furuncle. It may resemble certain forms of mastoiditis. (For differential diagnosis, see Mastoiditis.)

Prognosis.—The disease may get well leaving the canal practically as before, or it may pass into a chronic state resulting in a greater or less degree of stenosis of the meatus.

Treatment.—The treatment consists in irrigating the ear with warm antiseptic solutions, or packing the canal with gauze saturated with 4 to 8 per cent. solution of aluminum acetate. In severe processes which persist notwithstanding these measures, free incision of the canal walls becomes necessary. Several longitudinal cuts should be made through the soft parts to the bone

and cartilage, after which hot irrigations or the gauze packing should be resorted to. Frequently, the application of moist heat to the auricular region gives relief from the pain and seems to have a beneficial effect upon the process.

OTOMYCOSIS.

Otomycosis may be considered as a form of otitis externa due to the growth of fungi or mould in the meatus.

Causation.—There are a great many fungi capable of producing this disease, but those most frequently found

FIG. 43.—Aspergillus (Burnett). *a*, aspergillus glaucus; *b* and *c*, different stages of the growth of the sporangea of the aspergillus nigricans.

are the varieties of the aspergillus—niger, flavus and fumigatus (Figs. 43 and 44). All of the reasons for their being able to grow in some patients and not in others are not known. Oleaginous preparations favor the growth of certain varieties. These preparations are very popular with the laity, still otomycosis cannot be considered a common disease. Dampness seems to favor other forms. Ceruminous masses are frequently found to contain these fungi.

Symptoms.—In some patients considerable masses of these fungi may be in the ear without noticeable inconvenience. In others reaction of the skin takes place and

pain and pruritus may be produced. There may also be a discharge of watery material. Still in a great many instances the ears are dry. It is when the mould invades the tissues, either of the canal or the drum membrane, that symptoms are caused. If the growth produces a perforation of the latter structure, as they have been known to do, or if one exists, otherwise formed, the fungi may invade the tympanum, causing an otitis media. Upon inspection of the canal the appearance varies with nature of the fungus and the location of its growth. In the aspergillus niger the growth is covered with dark

FIG. 44.—Aspergillus (Burnett). Mycelial web before sporangea have developed.

spots, the sporangea, while in the flavus the masses are of a brownish-yellow color. They may be found upon the canal walls or upon the membrana tympani. The superficial layers of the skin exfoliate, leaving a moist surface. Sometimes the masses consisting of fungi and epithelial scales accumulate in sufficient quantity to occlude the lumen of the canal. In such instances there will be diminution of hearing, perhaps tinnitus as well.

Diagnosis.—The diagnosis is made from the characteristic appearance of the masses and the results of the microscopic examination of some of the material removed.

Prognosis.—The prognosis as a rule is good. It will usually be possible to cure the disease which they cause by destroying the vitality of the fungi.

Treatment.—In the treatment of this disease alcohol is a sovereign remedy. It is to be used as strong as the patient is able to bear it; beginning with 50 per cent. and increasing until pain is produced or 95 per cent. strength is reached. Bichloride of mercury 1 to 2000 in the alcohol adds materially to its action. The canal is first cleared of the growth as far as possible either by syringing or with the applicator, then after drying the parts the external canal is filled with the alcoholic solution, which is allowed to remain ten to twenty minutes. The cleansing of the canal need not be repeated unless found necessary, but the use of the alcohol should be repeated from two to four times a day until cure is complete.

OTITIS EXTERNA CIRCUMSCRIPTA.

Furuncle.

Causation.—Circumscribed external otitis or furuncle is a very common condition and is due to the entrance of bacteria into the glands of the external meatus, frequently after an abrasion. These abrasions are commonly produced by scratching the ear with the finger or some instrument which the patient uses, or they may result from injury inflicted while removing cerumen or a foreign body. The prevailing organism from pus taken from furuncles is the staphylococcus, although a circumscribed inflammation of the canal is occasionally produced by other germs, usually when it is secondary to middle-ear suppuration. Lack of resistance on the part of the patient is also, without doubt, frequently a factor in causation. The ceruminous glands and hair follicles are found only in the outer part of the canal. This makes a furuncle near the drum membrane an impossibility. A circumscribed inflammation at the fundus of the canal

is therefore to be considered as evidence of mastoid or middle-ear involvement. Furuncles are rare in young children.

Symptoms.—Circumscribed otitis externa usually begins with a stinging sensation, followed by soreness or pain upon moving the auricle or pressing upon the external auditory canal. Soon, if the infection is severe, there will be spontaneous pain. As the swelling increases and the canal becomes occluded, there is deafness and at times tinnitus. Furuncles may be divided into the superficial and deep varieties. In the first, the swelling is small and red and very soon shows a pustular summit. In the second, the swelling is larger, the redness is slower in appearing and considerable time (several days to a week) elapses before the skin breaks down, so that in some instances, the resistance being less in this direction, the pus may break through the canal wall and form an abscess adjacent to the auricle. In the superficial variety the abscess is often evacuated spontaneously without marked symptoms or swelling. In the deep, the pain and tenderness are more severe. If the furuncle is situated on the anterior canal wall, there is frequently pain, during mastication, from the condyle of the mandible disturbing the inflamed tissues. There may be edema of the tissues around the auricle. Fever is an occasional symptom but rarely a marked one. The rise of temperature is not over one or two degrees. If it is more than this in a patient supposed to have furuncle, the diagnosis should be carefully investigated.

Diagnosis.—At times the diagnosis of furuncle presents no difficulties. By inspection one sees a well-defined swelling within the meatus. Pain is elicited upon movement of the auricle or by pressing upon the swollen area. Careful introduction of the speculum demonstrates that the membrana tympani is normal as to color and luster and that the fundus of the canal is not contracted. There is nothing for which this picture can be mistaken. On the other hand, in patients with occlusion of the meatus,

with retro-auricular edema, the process may be mistaken for mastoiditis. If by using a small speculum a view of the fundus can be obtained and there is normal or nearly normal membrana tympani with no narrowing of the canal near the membrane, pain being present upon moving the auricle or canal and absent upon pressure over the mastoid, the process is one of furuncle. In examining the mastoid for tenderness in these patients great care should be taken that the thumb does not touch or move the auricle or external auditory canal, as this always produces pain in furuncle. When there is involvement of the middle ear and mastoid and the pus has reached the posterior wall either by dissecting its way out through the Rivinian segment or through the mastoid by a perforation on the posterior osseous canal wall, the resemblance to furuncle is quite marked. This subperiosteal accumulation of pus produces a swelling in the superficial part of the meatus and if it ruptures or is incised there is a discharge. Both of these conditions are apparently characteristic of furuncle. As there is a great difference in the treatment to be adopted, the differential diagnosis becomes of importance. Reliance is to be placed upon the following: (1) pain on movement of auricle present in furuncle, almost certainly absent in mastoiditis; (2) narrowing of canal near outer end in furuncle; at inner end contiguous to membrana tympani in mastoiditis. It is evident that if pus has dissected its way out beneath the attachment of Shrapnell's membrane the narrowing will extend to the fundus. If it reaches the canal through a perforation in the posterior bony wall, there is apt to be narrowing at the fundus, from mastoid involvement; (3) in furuncle the membrana tympani should be normal or nearly so. In acute purulent otitis media and mastoiditis the characteristic changes will be present; (4) if the abscess is opened the probe does not encounter bare bone in furuncle, except in rare instances. In mastoiditis, as the pus is subperiosteal, bare bone is always present or the probe may enter the mastoid through the

perforation; (5) pus from a furuncle usually contains the staphylococcus and is less in amount. In mastoiditis this germ is not so common and the discharge is more copious; (6) finally the *x*-rays may give valuable information in doubtful cases.

Treatment.—Some small furuncles may be allowed to pursue their course without treatment. Others if seen early are relieved by packing the canal with gauze saturated with a 4 to 8 per cent. solution of aluminum acetate. A narrow strand of gauze, first dipped into the solution, is then packed into the canal with a moderate degree of firmness and the patient is supplied with the solution and a few drops are occasionally placed upon the gauze to prevent its becoming dry. When there is severe pain, an early and free incision is the best treatment. If the swelling is of an oval form and well marked, the incision is made through the most prominent part, in the line of the canal and down to the cartilage or bone. If the prominence of the swelling cannot be made out readily, the point of greatest tenderness is ascertained by pressure with a cotton-tipped applicator and the longitudinal incision is made through it. If more than one tender point exists, an incision is made through each one. If the furuncle is a large one and has broken down, a strip of gauze may be inserted into the cavity for drainage. This is removed in twenty-four hours and another inserted if necessary. If the abscess is small or incised early, the canal may be packed with aluminum acetate gauze or syringed with bichloride or boracic acid solution. Packing the canal with gauze seems to more quickly relieve the swelling which frequently persists even after a free and sufficient incision. If the abscess has been neglected and points externally a counter opening may be necessary to insure sufficient drainage. This may be made freely, bearing in mind the anatomical structures to be encountered, gauze inserted and a moist dressing, either of aluminum acetate or saline, applied. At times as furuncles are healing, exuberant granulations, very much

resembling aural polyps, develop. These are to be removed with the sharp curette and their bases cauterized with the fused bead of silver nitrate. To prevent the occurrence of successive furuncles, an effort to prevent reinfection is made. Perhaps the best means to accomplish this is the instillation of the alcohol and bichloride solution. The patient is also cautioned as to scratching the ear with pins, ear spoons or other objects.

The internal treatment of furunculosis must not be neglected. Formerly great reliance was placed upon the compound syrup of hypophosphites which was much superior to the sulphide of calcium treatment once in vogue. The success attained by the employment of an autogenous vaccine is so great that it has supplanted other forms of general treatment. In every instance, when the incision is made, if pus is present, a culture should be taken so that, should a subsequent furuncle develop or the one incised be slow in healing, the vaccine may be at hand and administered.

EXOSTOSIS.

Bony growths from the osseous wall of the meatus are called exostoses. There may be either a diffuse osseous thickening or the new formation may exist as a well-defined tumor. Formerly the diffuse growths were called hyperostoses, but it is customary at present to make no distinction but to include them all under the term exostoses. These enlargements may be multiple or single, may be limited to one side or be bilateral. They may occur at the entrance of the osseous portion of the meatus or near the membrana tympani. There is no part of the bony canal that is exempt from them.

Causation.—Various causes have been assigned for the formation of exostoses but little is definitely known. There is no doubt but that a chronic suppuration of the middle ear may act as a causative factor. The author has observed two patients in whom there was involvement

of the anterior canal wall in leontiasis ossea. In one enlargement of the mandible occurred on both sides, but the exostosis was unilateral, while in the other (Fig. 45) the mandible was enlarged on one side only, that of the exostosis, and there was also a suppurative otitis media on this side. They have been found with uncommon frequency in Peruvian skulls and in those of the mound builders.

Fig. 45.—Patient with leontiasis ossea who had exostosis on the anterior wall of the external meatus.

Symptoms. — When symptoms are present, they are caused by the growths attaining sufficient size to obstruct the lumen of the canal or by the same result being accomplished by the retention of secretions internal to them. So long as the growths are small the patient may be unaware of their presence. One sees these exostoses from time to time in patients who have sought advice for some condition totally unconnected with them. As long as there is an opening in the canal and no secretion accumulates, these patients seem to hear about normally. When the patient comes on account of impaired hearing, tinnitus or a stuffy feeling in his ears, due to the disease, upon examination the growth or growths are found more

or less completely blocking the canal or the meatus is found narrowed by diffuse thickening. The probe shows them to be hard, resistant and not tender. They may have a broad base or this may be constricted so that they appear somewhat pedunculated. If the canal is blocked in a patient having a suppurative otitis media, there will be symptoms due to the lack of drainage and any of the complications given under Purulent Otitis Media may arise. When no symptoms are produced, they are usually discovered accidentally and either project into the canal as one or more small nodules or as an elevated area leading to contraction but not occlusion of the meatus. Exostoses at the junction of the fibrous and osseous portions of the canal are more apt to produce symptoms than those near the membrana tympani, as they lead more readily to the retention of cerumen, epithelial débris, etc.

Diagnosis.—The diagnosis as a rule presents no difficulty. The growth is seen and the probe shows it to be hard and firm. If the skin covering the exostoses is inflamed, thickened or ulcerated, there may be doubt but this would be dissipated by the use of the probe. To determine whether or not an otitis medis purulenta is present may be difficult. One would rely on the character of the discharge, if there was one or if drainage was completely blocked, the occurrence of symptoms pointing to some complication of middle-ear suppuration. In exceptional instances the question may not be settled until operation.

Prognosis.—Exostoses may be removed and they do not recur. There is, however, considerable difficulty in maintaining a free and sufficient meatus. There seems to be a special tendency for the parts to contract after the removal of exostoses. Either the bone becomes thickened or cicatricial tissue forms before the wound becomes dermatized.

Treatment.—If the exostosis is of small size, it requires no treatment. The patient should be seen from time to

time and the secretions removed. If it is so large that this becomes difficult or if it occludes the lumen of the canal, its removal by operation is indicated. If it leads to the retention of secretion in a suppurative otitis media, operation and the establishment of drainage is imperative. An exostosis with small peduncle may be removed with the gouge through the natural opening, but growths offering a prospect of success by this method are rare. Operation by a postauricular incision is usually the best, as it leaves a better meatus. An incision to the bone behind the auricle is made. The soft parts are elevated from the bone and the skin lining the meatus is carefully separated until the exostosis is exposed. This is then removed with the gouge, taking care not to injure the drum membrane. As much bone as possible is removed with the growth. The amount of this will vary with its location. If situated upon the inferior, superior or anterior wall, less, as a rule, can be taken. It is a good plan in this case to remove as much of the posterior wall as possible to increase the size of the meatus, that it may be sufficiently large notwithstanding subsequent contraction. If the exostosis is situated on the posterior wall, the bone removal is done with as little disturbance to the other walls and their cutaneous coverings as possible. If there is a suppurative otitis media, operative procedures for its relief may be combined with the removal of the exostosis. A flap is now cut in the meatus and concha, as described under the radical operation. It is preferable at this stage to cover all of the denuded bone with Thiersch grafts. If these take, the packing is removed in four days and the cavity cleansed by syringing, then wiping with peroxide, one-half strength of the 10-volume solution, followed by alcohol and bichloride 1 to 2000. If the graft does not take or has not been inserted, the canal should be firmly packed to keep down granulations. (See Care of Radical Cavities.) The posterior incision is closed by clamps or sutures and usually heals promptly.

STRICTURES AND ATRESIA OF THE MEATUS.

Contraction and occlusion of the meatus has been considered as producing symptoms in the various diseases of the external ear, the condition passing away with the cure of the disease. Patients present, however, in whom the contraction or atresia is a diseased condition by itself.

Pathology.—A stricture is a contraction of the lumen of the meatus, while an atresia is a total obstruction. The strictures are usually a result of an inflammatory process, with formation of new connective tissue, in the canal walls. If while the process is active and the canal walls are in contact abrasion of the opposing surfaces occurs, they adhere and an atresia is the result. If this adhesion is limited in extent a diaphragm-like membrane may follow; if more extensive, a thicker atresia is produced. One type of narrowing of the canal is found in old people and occasionally after the mastoid operation. The walls of the cartilaginous portion of the canal lie in contact so that sound waves cannot enter, and also allowing secretion to be retained. This type is due to atrophy of the cartilage in the canal walls so that it no longer serves to hold them apart. If the speculum is introduced, the canal remains open while it is in position but falls together when the speculum is removed.

Symptoms.—Strictures only produce symptoms when they lead to retention of secretions. The natural moisture of the skin of the canal internal to them being retained macerates the epithelium. This often decomposes and becomes foul-smelling. This material blocks the meatus and leads to deafness and perhaps tinnitus. As long as a small opening remains through the stricture and there is no moisture internal to it the patient hears very well, a very small opening being sufficient, apparently, to conduct the sonorous vibrations to the drum membrane. In atresias, as not even a small opening remains, there is always some impairment of hearing, the degree depending upon the thickness of the obstructing membrane.

When this is diaphragmatic and thin, deafness is not marked, unless secretion has accumulated internal to it. If the atresia is a thick one, and especially if it extends to the drum membrane, impairment of hearing is profound.

Diagnosis.—The diagnosis is usually easily made. Membranous atresias near the drum membrane may resemble this structure. The absence of landmarks and the fact that the canal is not as deep as normal, establishes the diagnosis. Comparison with the opposite side may aid one to determine their nature.

Treatment.—When there is no accumulation internal to the stricture, no treatment is necessary. If an accumulation is present, it is to be removed and the parts dusted with boracic acid. If there is middle-ear suppuration, a stricture interferes with treatment and an operation may be necessary. A thin diaphragmatic atresia may be incised and as much as possible of the membrane removed. For the thick atresias an operation, performed in a manner similar to the operation in exostosis, is necessary. The thickened walls are to be dissected out and the meatus enlarged by removing the posterior osseous canal wall. The cavity lined with grafts and a conchal flap made as in the radical operation.

In the type of stenosis occurring in old people and after the mastoid operation, a rubber tube may be worn, or a plastic operation to form a large meatus may be undertaken.

SEBACEOUS CYSTS.

Sebaceous cysts are of common occurrence on and around the auricle. Their favorite location is on the posterior surface of the lobule, although they may occur on any part of the auricle or even in the meatus. They are due to occlusion of the orifice of a sebaceous gland with the consequent retention of its secretion. They usually give rise to no symptoms unless located in the meatus, or they become inflamed. If a sebaceous cyst is removed entire together with its wall, it will not recur.

Treatment.—Two methods of treatment may be adopted. Either they may be incised, the cyst cavity evacuated, its walls curetted, then packed with gauze so that healing from the bottom takes place; or the cysts may be dissected out. The latter plan is better, leaving less cicatrix. An elliptical incision is made and the sac carefully separated from the surrounding tissue. If rupture occurs, the entire sac is to be removed. The wound heals with a linear scar which is scarcely noticeable.

TUMORS OF THE EXTERNAL EAR.

The external ear is subject to all of the tumors, both malignant and benign, which may occur in cutaneous, fibrous or cartilaginous structures in other regions, but as they are not of special otological interest, the student is referred to books on general surgery for their study.

CHAPTER IV.

DISEASES OF THE MEMBRANA TYMPANI.

The membrana tympani, dividing as it does the middle from the external ear, is common to both of these parts. Its external layer is continuous with the epithelial lining of the external auditory canal, its internal layer is a modified mucous membrane continuous with that of the middle ear, while between the two is the lamina propria, formed of connective tissue. This structure, consisting of epithelium, connective tissue and mucous membrane, is naturally subject to all of the diseases that affect these tissues. While almost all of the pathological processes which are observed in the membrana tympani are secondary to disease in the external auditory canal or middle ear, still occasionally disease develops in the structure as a primary process.

ACUTE MYRINGITIS.

Acute myringitis is an acute inflammation of the drum membrane and the term is used to apply to primary processes in this structure. If the epithelial layer is involved, there is an effusion and the formation of one or more blebs, which usually contain serum. If the fibrous layer becomes inflamed, an abscess may result which may rupture externally or into the middle ear. In the latter event an acute otitis media will result. Inflammation of the mucous layer is conceivable but it could not be recognized clinically as a process independent of otitis media.

Etiology.—The causes of acute myringitis are the application of irritating substances, mechanical irritation, cold

in the form of air or snow, and blows upon the ear not sufficient to cause rupture of the membrane, or the cause may be undiscoverable.

Symptoms.—There is pain, usually not as severe as in the acute inflammations of the middle ear. There may be slight tinnitus and some impairment of hearing. The symptoms vary in intensity with the severity of the process.

Diagnosis.—If one or more blebs are seen on the membrana tensa, the membrane otherwise being nearly normal, and inflation demonstrates that the middle ear is dry, one is justified in considering the process as myringitis. If the process clears up in a few days with return to normal hearing, without involvement of the middle ear, the correctness of the diagnosis is established. A small abscess in the fibrous layer would cause greater difficulty in diagnosis. If the swelling is circumscribed, the membrane otherwise nearly normal and the middle ear is dry, it would be fair to assume that the condition was one of myringitis affecting the lamina propria. However, the differential diagnosis from otitis media may be impossible. It must be remembered that the primary inflammations of the drum membrane are very rare, while inflammations of the middle ear are very common, and this fact should have due weight in making the diagnosis.

Prognosis.—Acute myringitis usually runs its course in a few days but it may become chronic or rupture may take place into the tympanum, resulting in otitis media purulenta.

Treatment.—The blebs may be opened, allowed to drain and boracic acid insufflated. This forms a crust which falls off in a few days. In patients who do not have these blebs but in whom the process is evidently mild in nature, irrigation with warm bichloride solution will relieve the symptoms and aid recovery. When there is a circumscribed thickening in the membrane, indicating abscess formation, as shown by its yellow color,

an attempt may be made to incise it without perforating the membrane. If this is impossible, the membrane should be freely incised and the patient treated as for otitis media purulenta acuta.

CHRONIC MYRINGITIS.

Chronic myringitis is usually a result of the acute form, and is due to the failure of the epithelium to cover the raw surface left after the rupture of a bleb or abscess in the membrane. This leaves an ulcer which becomes covered with granulations, or at times a small polyp.

Symptoms.—There is regularly a slight discharge. The patient may complain of tinnitus, slight deafness and an uncomfortable feeling in the ear.

Diagnosis.—The diagnosis is made upon the appearance of the membrana tympani which presents either a granulating surface or a small polyp, which is not accompanied by suppuration of the middle ear.

Treatment.—The indications for treatment are to remove the granulations or polyp and bring about dermatization of the surface. After anesthetizing the parts, the fused bead of silver nitrate is lightly applied; after which the area is dusted with boracic acid. If this fails to remove the polypoid tissue, it may be taken with the sharp ring curette. In cauterizing and curetting these areas care should be exercised to avoid perforating the membrane.

TRAUMATIC RUPTURE OF THE MEMBRANA TYMPANI.

Causation.—Rupture of the membrana tympani may be produced by direct injury to the membrane, by a foreign body or, indirectly, by a change in the air pressure in the external auditory meatus or tympanic cavity. Of those ruptured by direct violence, perhaps the pernicious habit, which is far from uncommon, of scratching

the ear with hair pins, ear spoons and various other devices, is responsible for the greater number. Next in frequency come the attempts to extract a foreign body which result in forcing it against the drum head, producing rupture. Rarer causes are bullet wounds, sword wounds, injury from pieces of steel, etc. Of those caused by a change of air pressure, the most frequently seen are due to a blow upon the ear which condenses the air in the meatus and forces the drum membrane inward until rupture takes place. Less common causes are falls upon the ear, explosions, or the increased air pressure encountered by caisson workers and divers. If the membrane is atrophied, it conduces to rupture. If the Eustachian tube is occluded or contracted, those causes which act through the surrounding atmosphere produce a greater effect upon the membrane. Ruptures from increased air pressure within the tympanum usually are caused by inflation and are considered under that subject.

Symptoms.—When rupture takes place the patient usually experiences the sensation of a loud noise. This may be followed by vertigo and faintness. Tinnitus and mild impairment of hearing may be present. The patient is aware that something is wrong with the ear and seeks advice to ascertain its nature. Occasionally there is bleeding, but this is not a common symptom. If infection of the middle ear occurs, there will be a purulent discharge and symptoms of acute suppurative otitis media.

Diagnosis.—The diagnosis is made from the history and by seeing the perforation. There is a great variety in shape and size of these ruptures, but usually no great difficulty is experienced in locating them. If one is in doubt auto-inflation or inflation with the catheter leads to the production of a whistle or blowing sound, according to the size of the opening.

Prognosis.—Those ruptures caused by condensing the air in the meatus or tympanum usually heal without noticeable impairment of function. Those produced by foreign bodies not infrequently result in middle-ear

infection and the prognosis becomes less favorable; although the suppurative processes following infection through traumatic ruptures of the drum membrane are usually of milder nature than those which result from infection through the Eustachian tube.

Treatment.—The ruptures which follow condensation of the air in the meatus or tympanum require no treatment as a rule. No harm occurs from syringing the ear with bichloride solution 1 to 4000, and this treatment may be adopted to prevent infection. These irrigations are of decided value in perforations caused by direct injury. One feels that in these infection is imminent if it has not already occurred. When the opening in the drum membrane is healed, if impairment of hearing remains, inflation may be of value.

Medicolegal Considerations.—A few weeks after the membrane is healed in many instances it is quite impossible to determine from the appearance of the drum head, not only the location at which rupture occurred, but also whether a rupture has been present. So if a patient claims to have had a traumatic rupture of the drum membrane, the surgeon cannot determine, in many instances, whether or not the claim is founded on fact. If there is no evidence that a perforation has been present, it does not prove the patient wrong in his claim. If this evidence is found, it is of value only insofar as that it establishes that a perforation was present but does not indicate its nature.

CHAPTER V.

NON-SUPPURATIVE DISEASES OF THE MIDDLE EAR.

In classifying the non-suppurative diseases of the middle ear and Eustachian tube, considerable difficulty is experienced. No classification is perfectly satisfactory. They will be discussed from a clinical rather than a pathological standpoint, as this will be less confusing to the student and give him more quickly a working knowledge.

TUBAL CATARRH.

Anatomical.—The Eustachian tube connects the nasopharynx with the middle ear. It is about one and one-half inches in length. Its outer half-inch is osseous, and the remaining portion is composed partly of cartilage and partly of fibrous tissue. In a cross-section of this part of the tube (Fig. 46), the cartilage appears as a hook, it being present on the posterior and upper walls. It thus reinforces about one-half of the tube and prevents its walls from collapsing when acted upon by some external force tending to separate them and thus open its central canal. The tensor and levator palati muscles are in relation with and attached to the tube in such a manner that during the act of deglutition when these muscles contract, the tube is opened and air from the nasopharynx readily passes into the tympanic cavity. This is nature's mechanism for maintaining that equilibrium of air pressure on the two sides of the drum membrane which is essential for its free vibration to occur.

Causation.—The disease may exist in an ear otherwise normal or may occur as part of an exacerbation in chronic

catarrhal otitis media. The disease is usually caused by some catarrhal condition in the nasopharynx. It occurs with colds in adults and with adenoids in children. Any inflammatory process in the nose or nasopharynx may cause it. Sea bathing is responsible for some cases. It may complicate any of the infectious diseases, although the aural involvement under these circumstances is apt to be of a more severe type.

Pathology.—In tubal catarrh the mucous membrane is swollen and contracts the tubal lumen. There is no doubt but that swelling and congestion of the tubal mucosa is

FIG. 46.—Cross-section of Eustachian tube, showing disposition of cartilage in the form of a hook. (Zuckerkandl.)

the first stage of many middle-ear processes. In an infective process of the tympanum, the infection usually travels along the Eustachian tube and an inflammation and swelling results. In this form of tubal involvement, the disease of which it is merely an incident, quickly produces symptoms and signs which overshadow those caused by the process in the tube. One thinks of the tube as being the avenue of infection, but as not being largely concerned with the clinical manifestations. When a non-infective process invades the tube it may go no farther, the middle-ear condition being produced by the mechanical effect

of tubal stenosis, in which event there exists the tubal catarrh under discussion. In calling this process non-infective, it is desired to signify that an absence of pyogenic microörganisms in sufficient number to produce pus is meant. It is not known but that some milder form of germ may be the cause of the tubal catarrh, or the swelling may be a congestion, perhaps also a collateral edema resultant from a neighboring inflammation in the naso-pharynx. The swelling and stenosis of the tube prevents the interchange of air between the nasopharynx and the tympanum, and the air in the middle ear becomes absorbed. It is easy to see how the oxygen in the air may be taken up by the bloodvessels, the tympanum acting in a manner similar to a pulmonary alveolus. But the condition present in many instances leads one to believe that much more than the oxygen content of this intratympanic air is absorbed. However, the air pressure within the tympanum becomes minus and the pressure on the outside of the membrana tympani remaining at that of atmospheric air causes the membrane to become depressed, and lose its ability to vibrate freely. This produces the signs and symptoms of tubal catarrh.

Symptoms.—The onset of the symptoms may be quite sudden or more gradual. The patients usually complain of a feeling of fulness or stuffiness in the ear, as though the external auditory canal was occluded. Some patients, who have had previous experience with impacted cerumen, come, asserting that they believe that they have a recurrence of this condition. At times there is tinnitus generally of mild degree, although it may be severe and overshadow all of the other symptoms. There is regularly impairment of hearing which functional tests show to be due to a lesion of the sound-conduction mechanism. The elevation of the lower-tone limit is not as a rule marked. The upper-tone limit is rarely affected. The tuning-fork tests show an increased bone conduction, a diminished positive, rarely a negative Rinné, with Weber usually lateralized to the involved ear if the disease is unilateral. In some patients

there are labyrinthine symptoms, chiefly vertigo, due no doubt to increased tension of the labyrinthine fluid caused by the stapes being forced into the oval window. Some patients are so annoyed by the feeling of pressure in the head that mental effort becomes impossible. There can be no doubt but that in children with adenoids this disease may become a factor in producing their lack of mental vigor.

Signs.—The tympanic membrane is normal in color, luster, integrity, and structure. Frequently there is injection of the mallear plexus showing as a red streak along the manubrium. The position of the drum head is altered. It will be found more or less depressed according to the duration of the attack and the elasticity of the membrane. In some instances the depression of the membrane is so extreme that it lies in contact with the internal tympanic wall. The depression will be readily appreciated by the general appearance of the membrane, the foreshortening of the manubrium and by the prominence of the posterior fold and tympanic ring.

Diagnosis.—The diagnosis is made from the fact that the patient has a feeling of fulness in the ear, tinnitus or deafness; that these symtoms have been of short duration; that examination shows a depressed drum membrane otherwise practically normal, and that the middle ear is dry, with perhaps other characteristics of the inflation sound described under Treatment.

Prognosis.—Some tubal catarrhs get well in a few days or weeks without apparent effect on the function of the ear, others take much longer. Some develop tympanic congestion, while others pass into a chronic state and become a factor in the development of chronic catarrhal otitis media.

Treatment.—The treatment consists in catheterization and attending to the condition of the Eustachian tube and nasopharynx. Upon inflation the tympanic membrane frequently goes back into place with a distinct snap or thud. If this occurs upon the first gentle pressure

of the bulb, it is fair to assume that the tubal swelling is at the pharyngeal orifice, and that the point of the catheter has passed beyond it. If the tumefaction is farther along the tube, air may not pass through until after the bulb has been compressed two, three or even more times. Its passage may then he accompanied by a high-pitched note, at times a whistle, subsequent blasts requiring considerable pressure on the bulb to force air through. At times inflation seems to dislodge a plug of tenacious mucus. When this is the case, subsequent air blasts are more free. Inflation is to be repeated daily or every second day until the tube remains open. Usually immediately after inflation the patient experiences considerable relief, exceptionally he feels as bad or slightly worse. This will be due to the fact that, owing to the narrowness of the tube, the air forced into the tympanum cannot return, and there is thus produced increased pressure with bulging of the drum membrane. This will pass away in an hour or so with a sensation of relief from pressure and improvement of hearing. Applications of argyrol, 25 per cent., or silver nitrate, 2 to 4 per cent., are to be made to the mouth of the tube and nasopharynx. The use of cocaine for these inflations seems to diminish tubal engorgement, acting directly when applied to the tubal orifice with the applicator, and indirectly through its action on the vessels of the inferior turbinate which are intimately connected with those of the tube. In stubborn cases applications of cocaine followed by argyrol or nitrate of silver solutions may be made directly to the tubal mucosa, by inserting a cotton-tipped applicator through the Eustachian catheter. These applicators may be bought in the shops, or a very useful one may be made from No. 5 piano wire, as suggested by Dr. Dench.[1] The wire is roughened at the end so that the cotton may be firmly retained. It is then passed through the catheter, which is to be used, until it projects one and one-half

[1] Diseases of the Ear, third edition, p. 313.

inches beyond the tip and the wire bent at a right angle at the ring end. This gives the measurement beyond which it is not safe to pass the applicator. Having inserted the catheter and ascertained that it is in position, the applicator, armed with a small and firmly wound cotton tip, which has been saturated with the desired solution, is passed through the catheter into the tube. The patient is requested to swallow as any obstruction is encountered, as this opens the tube and allows the applicator to be passed more easily. In patients showing a tendency to repeated attacks of tubal catarrh, the nose and nasopharyngeal condition should be attended to, enlarged posterior tips of the inferior turbinate snared or any other abnormal condition corrected, and the adenoids should be removed in children.

TUBOTYMPANITES (TUBOTYMPANIC CONGESTION).

Causation.—The causation is the same as in tubal catarrh, as it usually follows the latter disease, especially if proper treatment has not been promptly instituted.

Pathology.—In tubotympanites, in addition to the changes in the Eustachian tube, characteristic of tubal catarrh, there is also congestion of the tympanic mucous membrane. This condition leads to a serous effusion or increase of mucus, so that it becomes evident upon inflation. The changes in the mucous membrane of the tympanum may be in the nature of a passive congestion. When this exists it may be fairly attributable to the diminished intratympanic pressure which has been brought about by the tubal occlusion. This venous congestion is followed by the transudation of serum, which may fill the tympanic cavity more or less completely. The condition of the bloodvessels associated with chronic renal, cardiac or hepatic disease, may aid in bringing about this result. In the second form the congestion in the mucous membrane of the middle ear is an extension of the process in the tube, and not directly caused by the

change in intratympanic air pressure and the effusion is of a more mucoid character. While these two types may sometimes be separated clinically, no attempt to do so will be made in the present discussion. After the mucous membrane in the tympanum becomes swollen and fluid transudes, the minus intratympanic pressure is relieved and the drum head may return to its original position. The swelling of the tubal mucosa or the accumulation of tenacious mucus within the tube may so interfere with the drainage of the mucus, or serum may accumulate in sufficient quantity to produce a mild increase in the pressure within the tympanum, but never to the same degree as in active middle-ear inflammation. In fact, the instances in which it is possible to recognize this increased pressure by signs and symptoms are uncommon and may be considered as on the border-line of acute catarrhal otitis media. The disease is usually of an acute or subacute nature.

Symptoms.—There is a feeling of fulness and stuffiness in the ear and discomfort in the head, but usually absence of pain at least severe in degree. Exceptionally, the patient describes his sensation as being one of pain, but upon careful inquiry it is found not to be like the severe pain of acute inflammation which keeps the patient awake at night. However, the dividing line between the acute type of tubotympanic congestion and the mild form of acute catarrhal otitis media may at times be hard to place as regards the pain. Still, pain even of mild degree may be considered the exception in tubotympanites. There is always some impairment of hearing, the degree depending to a certain extent upon the amount and location of the fluid. When this is excessive so that the oval and round windows are covered, the deafness is quite marked. When the fluid is less in amount, the impairment of hearing is often increased when the head is inclined backward or the patient lies on his back. In this position the fluid accumulates in the region of the labyrinthine windows. With the head inclined forward relief may be

experienced as the fluid gravitates away from the fenestræ. There is increase in bone conduction varying with the degree of the impairment of hearing. This gives rise to positive Schwabach and Weber tests, with a diminished positive, rarely a negative Rinné. The lower-tone limit is apt to be elevated but the upper-tone limit remains normal, unless there are labyrinthine changes which are not a part of this disease. A frequent symptom is a distressing click heard upon swallowing, due to the effect upon the fluid within the tympanum produced by nature's attempt to open the Eustachian tube. Autophony is at times an annoying symptom; the patient's own voice has an unnatural sound, as though he was talking with his head in a barrel. Tinnitus is also a common symptom. Some of these patients complain of a feeling as though something was moving in the ear. This is due, no doubt, to changes in the position of the fluid within the tympanum. There is usually no rise of temperature. If fever is present and due to the aural condition, one may feel quite positive that it should be classed as an acute inflammation of the middle ear and not as tubotympanites.

Signs.—Upon inspecting the membrana tympani the luster is usually normal. The color may be normal or of a yellowish tinge from the serum showing through, or of a dull bluish color. Upon closer inspection the latter color is found to be due to venous engorgement, the vessels at times showing quite plainly throughout the membrane. The manubrial and peripheral plexuses may be injected and red, but this bluish appearance of the membrana tensa is characteristic and is probably present in the early stages of this disease in the majority of instances. The engorgement of the drum head mucosa, to which this appearance is due, clears up more rapidly than that of the rest of the tympanum, so that when the patient comes under observation this appearance of the drum membrane may be absent, although it is not infrequently seen. When the fluid in the middle ear is considerable in amount, it is possible at times to determine its level which is marked

by a transverse line, and after inflation the membrane below this line may be studded with little spots in motion which are really bubbles in the intratympanic fluid. The student must not be disappointed if he sees this but seldom, as it is not usually noticeable. The drum membrane may be normal in position, slightly retracted or the retraction may be less than normal. Bulging is of very uncommon occurrence. While its possibility cannot be denied from the conception of the disease as above stated, still its occurrence would lead one to doubt that the process in which it was present really belonged to this class. Upon inflating with the catheter there is a bubbling, crackling sound either as the air is blown through or immediately following this, when the pressure upon the bulb is relaxed. This sound is heard as though located in the examiner's ear, and must not be confused with rales produced at the pharyngeal orifice of the tube, which gives one the impression as of coming from outside of the ear. Occasionally these will be present in addition to the intratympanic crackling, and are due no doubt to the presence of the fluid which has been forced out of the ear by the air blast.

Diagnosis.—The sound of crepitation or crackling from the intratympanic fluid is characteristic, and establishes the diagnosis if suppurative processes with fluid within the tympanum and the exudative form of chronic catarrhal otitis media can be excluded. From the former the diagnosis is made by the absence in tubotympanites of pain or fever, and the absence of the changes in the tympanic membrane which are found in the acute middle-ear inflammations. From the exudative type of chronic catarrhal otitis media the diagnosis may be extremely difficult. When the drum membrane presents the bluish appearance characteristic of the early stages of tubotympanic congestion, this establishes the diagnosis. The element of time may also be considered, this disease being essentially acute or subacute. The feeling of fulness or stuffiness in the ears is more often complained of in tubo-

tympanites, while in chronic catarrhal otitis media the patient more often complains of deafness. If tubotympanic congestion arises in a patient suffering from the hyperplastic form of chronic catarrhal otitis media, or having existed results in the exudative form, the diagnosis may be impossible with any degree of certainty. It is probable that there is no hard-and-fast line between the subacute form of tubotympanic congestion and the exudative form of chronic catarrhal otitis media, and as treatment is the same in either case it may be necessary to leave the diagnosis in doubt.

Prognosis.—This disease may result in cure with very slight or moderate deafness, or changes may take place in the mucous membrane of the middle ear of a more or less permanent nature, so that when the attack passes off and the ear is dry the patient may have chronic catarrhal otitis media of the adhesive or hyperplastic type, or the tubotympanities may become chronic, resulting in the exudative type.

Treatment.—The indications for treatment are to remove the fluid and to restore the mucous membrane as far as possible to a normal condition. These indications are best fulfilled by inflation performed every second or third day, followed by the application of nitrate of silver solution, 2 to 4 per cent., to the mouth of the tube and nasopharynx. In this disease the use of nitrate of silver seems to be of more value than argyrol; however, the latter may be applied through the catheter, as advised in tubal catarrh. Stimulating vapors will be found of value in these patients. The following formulæ are suggested:

℞—Tr. iodini . . . ʒss	℞—Menthol,
Chloroformi,	Camphor . . āā ʒj
Alcoholis . . āā ʒj—M	Tr. iodini . . ʒj—M

A few drops of one of these solutions is placed upon cotton contained in the rubber bottle attached to the inflating apparatus and the stopper turned so that, as the air passes through the bottle, it becomes impregnated

with the vapor of these volatile substances and thus carries the medication into the tympanum. In many patients after treatment, the condition clears up and the ear becomes dry. In some the process seems quite stubborn. At times the patient will cease attending for a few weeks and return with a dry middle ear. So if treatment produces no effect after a fair trial, a rest may be advised. As a last resort, the tympanic membrane may be incised, after which the fluid is driven out of the tympanum through the opening by inflation or through the Eustachian tube by the otoscope. It would seem that this procedure ought to give relief, but it seldom produces permanent improvement. The fluid is apt to reaccumulate, and the perforation heals, leaving the patient no better than before, or infection may occur and the disease become of a suppurative nature. It is possible that these patients are more susceptible to suppurative otitis media than others, and that infection through the tube may cause this condition. Some aurists believe that inflation may be a menace for this reason. Experience teaches that this belief is without foundation, as a careful inflation, skilfully performed, very rarely, if ever, gives rise to infection. Everything possible should be done to improve the general health and to relieve any abnormal condition of the nose or nasopharynx.

OTITIS MEDIA CATARRHALIS CHRONICA (O. M. C. C.) (CHRONIC CATARRHAL OTITIS MEDIA).

Chronic catarrhal otitis media, or O. M. C. C., as it is usually called by aurists, is a chronic affection of the mucous membrane of the tympanum and Eustachian tube. The change in the mucous membrane may be in the nature of an engorgement and consequent thickening with an excess of secretion, the exudative type, or there may be formation of connective tissue in the form of diffuse thickening, adhesions or bands. This is called the hyperplastic or adhesive type. While the division of

O. M. C. C. into the exudative and hyperplastic types is open to considerable criticism, still it answers very well and is about the only clinical division that can be made that is based upon the pathological conditions. The word "exudative" is used to indicate an increase of moisture within the tympanum without regard to the manner of its production. This, as will be shown, may be a transudation of serum, or an increased secretion of mucus, so in speaking of this type as an exudative catarrh, while the word is not strictly correct in a pathological sense, it is nevertheless convenient when used in this way by clinicians.

Exudative Type of Chronic Catarrhal Otitis Media.—The clinical characteristic of this type is a transudation of serum or the secretion of mucus on the surface of the tympanic mucous membrane in sufficient quantity to be detected upon catheterization. If the fluid is less in amount than this, there are no means by which this type may be separated clinically.

Causation.—The disease may originate from a naso-pharyngeal catarrh or may occur, when this is absent, without known cause. Chronic catarrh of the Eustachian tube is frequently responsible for it, or it may arise from tubotympanic congestion. Adenoids is an important factor in many instances. Nasal obstruction and enlargement of the posterior end of the lower turbinates seem to exert a causative influence.

Pathology.—The mucous membrane is swollen and engorged as a result of diminished intratympanic pressure, or from an extension of a similar condition in the Eustachian tube, or the process may originate in the mucous membrane of the tympanum. It is evident that there may be one of two processes in the mucosa: (1) a passive congestion with the transudation of serum or sero-mucus; (2) for lack of a better name, may be called a catarrhal process, which is accompanied by increased blood supply with thickening of the mucous membrane and increased production of mucus. These

processes are essentially chronic in nature, and may or may not be associated with hyperplastic changes; but if they continue to exist unrelieved, they are almost certain to result in the production of connective tissue in the form of diffuse thickening, adhesions or bands. Or the exudation may be developed in an ear which is already the seat of the hyperplastic form of O. M. C. C. However this may be, the fluid contents of the tympanum, while not necessarily causing the symptoms, enable one to establish this form of otitis media.

Symptoms.—Deafness is a constant symptom of this disease. The degree of impairment of hearing is not usually as extreme as in the hyperplastic type, unless the fluid exists in sufficient quantity to cover the oval and round windows, when it may be quite profound. While the deafness may be due to the fluid to a greater or less degree, it is at least partially caused by changes in the mucous membrane which, while it becomes less pliable than normal, never assumes the rigidity which is present after hyperplastic changes have taken place. There is an elevation of the lower-tone limit, an increased bone conduction, giving rise in persons under fifty to a positive Schwabach and Weber, with a negative or diminished positive Rinné. Subjective noises are apt to be a prominent symptom. The patient complains of roaring, rumbling sounds. They are usually of lower pitch than in the hyperplastic type. There may be a feeling of fulness in the head, and the patient is apt to be nervous and disinclined to mental effort. Autophony is sometimes complained of. Pain is not present, but the distressing feeling in the head of which these patients sometimes complain is no doubt harder to bear than acute pain would be.

Signs.—The drum membrane may be depressed. It may also appear to have a yellowish tinge from the fluid in the tympanum. This may also produce a line marking the fluid as described under Tubotympanites. The luster is usually normal and the structure not altered. The drum

membrane more often than otherwise is practically normal in appearance. Upon inflation the tube may be found more or less stenosed, either on account of the swelling of the mucosa or the presence of tenacious mucus. This is shown by the fact that the air passes less readily through the tube, requiring greater pressure upon the bulb, or only passing through upon the second or subsequent compressions. Before the air enters the middle ear an auscultation sound, due to its impact upon the mucus contained within the tympanum, may sometimes be heard. This is a clicking sound followed by a murmur which gives one the impression of coming from outside the inflator's ear. This element of distance shows that air does not enter and circulate within the tympanic cavity. As the air enters the middle ear, two varieties of sound may be interpreted as being caused by intratympanic fluid. The first when the fluid is small in amount and tenacious, which causes opposed surfaces of mucous membrane to adhere. As the air blast enters these are separated and a high-pitched sound is produced. It is of short duration and only heard upon the entrance of the air. The murmur that follows is usually dry and varying in pitch, with the lumen of the tube. The second occurs when the fluid is more considerable in amount and is a crackling, bubbling, crepitation sound which presents considerable variation in different patients. The sound occurs either while the bulb is being compressed or after relaxing the grip, and is due to air being forced through the fluid. A certain amount of knowledge concerning the character of the fluid may be derived from a study of these sounds. It is presumed that a considerable part of the crepitation is produced by the rupture of the bubbles which the air produces in this intratympanic fluid. This being so, the finer the crepitation sound, the less viscid the fluid, that is the more serous; while the coarse sounds, and especially those delayed until after the bulb is relaxed, indicate a greater viscosity of the fluid and therefore a greater mucus content.

Diagnosis.—The diagnosis of this type of O. M. C. C. from tubotympanic congestion, which it greatly resembles in many respects, has been discussed under the latter disease. That it is a form of O. M. C. C. is determined by the presence of chronic deafness, due to a lesion of the sound-conducting mechanism and not associated with any of the fundus changes of a purulent otitis media, or its residual processes. From the hyperplastic or adhesive type it is separated by the characteristic auscultation sounds. When the two processes are associated in the same patient, it may be impossible to determine how much of the deafness is due to the exudative and how much to the hyperplastic type. The only data from which one may draw a conclusion are the amount of improvement in hearing after inflation, which is usually greater in the former type, and the increase in hearing when by treatment the aural condition improves so that the characteristic auscultation sounds disappear, the greater the improvement, the greater the probability that the process was of the exudative type. These data, it must be conceded, are uncertain, as some thickening of the tympanic mucous membrane, not due to hyperplastic changes, may be left after the fluid has disappeared and be productive of a certain amount of impairment of hearing. When there is exudative catarrh and the fluid does not exist in sufficient amount to give the characteristic auscultation signs, or is so located that it does not do so, there are no means by which such a condition can be diagnosed from the hyperplastic type.

Prognosis.—The prognosis, while uncertain, is decidedly better than in the hyperplastic or dry form. Many patients are benefited by treatment. Those processes resulting from tubal obstruction are perhaps the most favorable. It must be remembered that the adhesive type may result from the exudative, and if the changes are in certain locations, marked deafness may result. So while encouraging the patient to hope for amelioration, the prognosis should be quite guarded.

Treatment.—In treating this condition the indications are to remove the fluid and to relieve the underlying condition in the tympanum and tube. The best way to meet these indications at present known is by inflation through the catheter. The fluid is, under favorable conditions, at least partially forced out through the tube; the increased intratympanic pressure produced by inflation tends to lessen the congestion; and the stretching of the tube has a tendency to diminish the engorgement of its mucosa. Inflation should be performed every two or three days, for a few weeks, then less often. No rule can be given, but the progress made will indicate when inflation should be done less frequently. The vapors, advised under tubotympanic congestion, have a favorable action both in stimulating the mucous membrane to absorb the fluid and in bringing it into a healthier condition. The mucous membrane in the pharyngeal orifice of the tube should be anesthetized with a solution of cocaine, or if some other anesthetic is used which does not produce contraction of the mucous membrane, it is to be followed by the application of one of the preparations of the suprarenal capsule, such as adrenalin, passed well into the tubal orifice to produce contraction, as far as possible, of the tubal mucosa. Then after inflation an application of 2 to 4 per cent. of silver nitrate is made well into the tubal orifice with a curved applicator. If deemed advisable on account of the persistence of the swelling of the tubal mucosa, as shown upon catheterization, applications of cocaine followed by argyrol or silver nitrate may be made throughout the length of the tube, as described under Tubal Catarrh. The injection of fluid through the catheter into the middle ear, as advised by some, does not appeal to the author as being a perfectly safe procedure. Tenacious mucus is the most difficult to deal with by inflation. An incision may be made in the membrana tympani and the fluid forced out through the opening. This may be successful in some instances, but too much must not be expected from this mode of treat-

ment. Any existing abnormality of the nose or nasopharynx should be relieved if possible. If adenoids are present, their removal may, and usually does, result in improvement. In other abnormal conditions which may be found, while their correction is the proper procedure, too much hope of relief of the aural condition should not be encouraged. One is continually being consulted by patients who have submitted to an operation on some nasal or pharyngeal condition, being encouraged thereto by the assurance that aural improvement would result, and who have been disappointed in finding that their faith has not been justified by the results. For the noises in the head which these patients experience, in addition to the treatment outlined above, the bromides in 15-grain doses, three times a day, are as good as any internal medication. The general health should receive attention and constipation relieved, especially if tinnitus is present.

Hyperplastic or Dry Type.—Adhesive Catarrh.—The hyperplastic or adhesive type of chronic catarrhal otitis media is a common disease and much more frequently seen than the form accompanied by fluid in the tympanum.

Causation.—The causes of O. M. C. C. are frequent attacks of tubal catarrh or tubotympanic congestion; catarrhal processes in the nasopharynx; hereditary predisposition, although if this is marked it favors the diagnosis of otosclerosis; frequent colds, bad climatic conditions and unhygienic surroundings.

Pathology.—The pathological condition is the formation of new connective tissue. This may occur in the form of diffuse or localized thickening, in adhesions between the various intratympanic structures or in bands. If any of these conditions exist and do not produce deafness, the notice of the aurist is not directed to them. In order to produce deafness some interference with the transmission of sonorous vibrations from the drum head through the ossicular chain to the labyrinthine fluid must be present, or there must be some loss of motility of the membrane closing the round window, as these are the avenues through

which sound reaches the labyrinthine fluid and perceptive mechanism. According to location, the lesions may be divided into four groups:

1. Those which interfere with the motility of the membrana tympani: (*a*) processes causing contraction of the Eustachian tube operating as in tubal catarrh; (*b*) adhesions between the drum membrane and the internal tympanic wall; (*c*) more rarely the drum head itself may be thickened and its motility thereby impaired.

2. Lesions interfering with the motility of the ossicular chain: (*a*) adhesions between the handle of the malleus and internal tympanic wall; (*b*) adhesions or bands between the malleus and incus and the tegmen tympani; (*c*) ankylosis of the joints between the malleus and incus or between the incus and stapes; (*d*) adhesions between the long process of the incus and the internal tympanic wall.

3. Lesions interfering with the motility of the stapes: (*a*) adhesions or bands between the crura and the walls of the niche of the oval window; (*b*) thickening of the mucous membrane around the foot-plate; (*c*) deposit of lime salts in the annular ligament.

4. Lesions interfering with the motility of the membrana tympani secondaria: (*a*) thickening of the mucous membrane; (*b*) bands and adhesions in the niche of the round window. There are great variations both in the location of the lesions and the degree of their development.

The tympanum contains many folds of mucous membrane, and for normal hearing their perfect pliability is necessary. Even a slight thickening and rigidity of these folds interferes with the proper transmission of sonorous vibrations.

Symptoms.—Deafness is a constant symptom. Its degree varies greatly. Some patients are unaware of its presence either because it is unilateral, or on account of its being so slight. The amount of hearing possessed by the normal ear is largely in excess of the requirements of daily life in the ordinary vocations; so the patient only

discovers that he is deaf when some extraordinary demand is made upon his hearing power, or when led by the occurrence of tinitus he seeks advice and it is discovered by the aurist. Others when coming under observation are profoundly deaf so that they cannot hear ordinary conversation even in their immediate proximity. Between these two extremes are all grades of deafness in patients presenting with the adhesive form of O. M. C. C. The deafness may progress slowly or remain stationary for periods of time. Some patients get worse only after attacks of tubal catarrh or tubotympanic congestion, each attack leaving them with a little more impairment of hearing, perhaps also more tinnitus than before, while during the attack the deafness is considerably increased. When one ear is involved the opposite ear is more apt to become affected than in a normal individual. When the deafness in one ear becomes quite marked and then the other ear becomes involved, it commonly occurs that the disease in the second ear progresses more rapidly than in the first ear to become affected, so in a varying time the ear last involved becomes the deafer of the two. A symptom sometimes present when the deafness is quite marked is known as paracusis Willisii. Patients with this symptom hear better in the presence of a loud noise. For example, while riding in the subway or in a noisy street they can hear conversation which in a quiet place would be impossible. The explanation of this symptom is that the intense sonorous vibrations produced by the loud noises, set in vibration the stiffened conductive mechanism and in this condition it more readily responds to the gentle waves of the conversational voice. There is regularly an elevation of the lower-tone limit, varying usually with the degree of impairment of hearing. The deafer the patient, the more the limit is elevated. Lowering of the upper-tone limit sometimes occurs from extension to the labyrinth. If this is at all marked it favors the diagnosis of otosclerosis. There is increased bone conduction becoming, if deafness is marked, greater than air

conduction (with 256 D. V. fork B. C. +, B. C. > A. C. Weber + to deafer ear).

The subjective head noises are of almost endless variety and intensity. They are, however, apt to be worse than in suppurative processes or even than in the other forms of non-suppurative middle-ear disease. These patients frequently complain of "shooting" pains in and around the ear. These pains are intermittent and not to be compared in severity with those which are produced by acute middle-ear inflammation. The strain under which patients with severe deafness labor is frequently very apparent both in their facial expression and in their manner. They watch one's lips closely, thereby being aided to a knowledge of one's conversation that the hearing alone could not give. Some patients are very sensitive about their infirmity and resent one's speaking to them in a loud voice. Many, no doubt, can hear better if the voice is not so loud, but the articulation distinct. Vertigo is sometimes a symptom and indicates that there is some disturbance of the labyrinth—a change in the labyrinthine structures or circulation; or in the pressure of the fluid within the labyrinth.

Signs.—The luster of the membrana tympani is either normal or but slightly diminished. The color is never red, but is normal or whiter than normal. The integrity is unimpaired. If any changes exist they are in the structure and position. These structural changes show as opaque spots or plaques or involve the whole membrane, or the membrane may be thin and transparent from atrophy, allowing the color of the pink mucous membrane on the internal tympanic wall to show through, and the long process of the incus articulated with the stapes to be seen. The drum head will be depressed if the Eustachian tube is stenosed or the membrane has been relaxed through stretching from former stenosis. The same result may be brought about by adhesions which bind the membrane or the handle of the malleus to the internal tympanic wall. One quite characteristic appearance of the drum membrane

is due to depressed areas. They at times very closely resemble old perforations which have been closed with thin cicatricial tissue. These areas are caused by atrophy of the membrane associated with long-continued minus intratympanic pressure. At times the cicatricial bands in the tympanic cavity have resulted in rotation of the malleus. If this ossicle is turned so that the short process points more forward than normal, the manubrium will appear broadened; if rotated so that it points more posteriorly than it normally does, it will appear narrower.

Inflation.—Inflation may show that the Eustachian tube is in any degree of patency. When very narrow, the tubal constriction is not as easily overcome as in acute tubal catarrh. It may require considerable force to overcome the obstruction, and the air enters the tympanum in small quantity with a rather high-pitched murmur throughout the inflation, although at times toward the latter part it may enter more freely with a somewhat lower-pitched murmur, showing that some of the constriction is due to engorgement of the tubal mucosa, although most of it is caused by hyperplastic changes. On the other hand, the tube may be of abnormal patency, giving a low-pitched murmur with the impression of abundance of air passing through the tympanum. The sound produced by vibration of the drum head may be a dull thud, showing that it is not noticeably relaxed; or there may be a sharp snap, showing relaxation of the membrane. This high-pitched snap is more often found when the depressed areas alluded to above are present. Considerable discussion has taken place in recent years as to the effect on the drum head of what has been termed "overinflation," some otologists claiming that this relaxed membrane which moves out with a distinct snap is due to "overinflation." While it cannot be doubted that frequent inflations by Valsalva's method may be harmful by causing a congestion of the head and ears, perhaps also some relaxation of the drum membrane, there appears to be very little in this "overinflation" theory

as the cause of these relaxed drum membranes. The author has under observation at the present time patients who have been inflated regularly for years, whose drum membranes are not abnormally flaccid, and others are constantly coming under observation who have never been inflated and yet present these relaxed membranes. The force producing them is not "overinflation," but long-continued minus intratympanic pressure, aided no doubt in many instances by atrophic or degenerative changes in the membrane itself.

Diagnosis.—The diagnosis of the hyperplastic or adhesive type of O. M. C. C. rests on the following considerations: The presence of chronic deafness which tests show to be due to involvement of the sound-conducting mechanism; the appearance of the drum membrane being practically normal in color, luster and integrity (for exceptions, see Signs); and by inflation demonstrating a dry middle ear. The diagnosis from otosclerosis will be considered under that disease, while the separation of this type from the exudative type has already been considered.

Prognosis.—The prognosis of this form of O. M. C. C. cannot be regarded as favorable insofar as the hearing is concerned. Some patients are improved to a moderate degree; others, perhaps to a certain extent on account of treatment, remain stationary, while many, no matter what treatment is adopted, become progressively worse.

Treatment.—The treatment cannot be regarded as satisfactory. While a great deal may be done for these patients and many improve, still in spite of all of one's efforts some remain stationary or even get worse. Those patients in whom involvement of the Eustachian tube increases the deafness generally experience most benefit. In this class it is usual to have increased hearing after inflation, or if the tube is so constricted that the air cannot return but produces increased pressure in the tympanum, the improvement occurs within an hour or so, as the pressure becomes equal on both sides of the drum membrane. In either event it is fair to assume that part

of the deafness is due to lack of motility of the drum head from minus intratympanic pressure, and improvement by treatment may be expected if the tubal condition can be relieved. An attempt to accomplish this is made by inflation repeated every three days, and the application of argyrol or silver nitrate to the mouth of the tube or to the tube itself after the inflation (see Tubal Catarrh).

In patients with marked tubal stenosis good results are often obtained from the use of whalebone bougies (Fig. 47). The catheter is first introduced and inflation performed. This establishes the fact that the catheter is in the mouth of the tube. Then the smallest-sized bougie is passed through the catheter to a depth of one and one-quarter inches. It is allowed to remain a few moments,

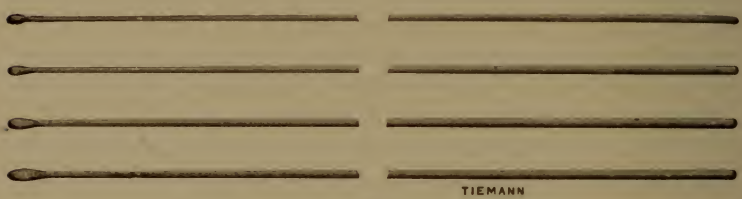

FIG. 47.—Author's whalebone bougies.

then the next size, and so on until the largest size that will pass through the constriction has been used. The patient is not to be inflated for three or four days on account of the danger of emphysema. This procedure may be repeated every ten days or two weeks, until the inflation sound shows improvement. It not rarely results in some relief from the tinnitus which may be present. In patients who have a complicating acute tubal catarrh or tubotympanites, the attack of these diseases is to be treated until relieved to prevent added impairment of hearing. To those patients who get worse by repeated attacks of these diseases, the aurist becomes of real value. He treats them through these attacks and preserves the hearing for years, in some instances without allowing much added impairment to take place. In patients in whom

the lesion is not in the tube very little or no improvement occurs after one inflation; nevertheless some of these patients will improve if inflated twice a week for a month and once a week thereafter. It may be found after several months that they are better. It is impossible to tell beforehand which ones will improve. Then in others it is possible that the disease is held in check by the massage of the middle-ear structures afforded by inflations. Patients are seen who with faithful treatment remain stationary for years, then becoming discouraged at their non-improvement, they cease to attend and grow worse rapidly. The number who give this history is too considerable to attribute it to chance. Vibrations and massage with various sorts of apparatus, such as the otoscope, Delstanche's masseur, electric and pneumatic apparatus, have been tried and recommended by some, but they are all of slight value as compared with inflation by the catheter. For the noises the bromides may be given a trial in addition to the other treatment. The care of the general health is not to be neglected. Various operations have been performed upon the ear with a view to increasing the hearing. Formation of an artificial opening in the drum membrane may produce temporary improvement in proper cases, but the opening usually soon closes and the condition becomes as before. The stapes has been removed, but the scar tissue forming in the niche of the oval window during healing is apt to result in greater impairment of hearing than before, if the patient escapes total deafness from labyrinthine inflammation. Removal of the malleus and incus together with the drum membrane has had its advocates. There can be no doubt but that temporary improvement may be produced by this procedure in properly selected patients, but in the majority of them during the healing process the mucous membrane becomes thickened and the patients' hearing returns to its former condition. These operations are quite fascinating, but to the conscientious surgeon are apt to be disappointing. Still, if the patient wants something more

done after stating the case to him the surgeon is justified in undertaking them. The operation of removing the malleus and incus for the relief of deafness is practically the same as when this operation is performed for the relief of chronic suppuration (see Ossiculectomy, page 269); the only difference being that there are no granulations to remove and that any bands or adhesions in the niches of the oval and round windows are to be incised.

OTOSCLEROSIS, OSTEITIS METAPLASTICA.

The term "otosclerosis" was applied to a form of deafness due to lesion of the conducting mechanism before the true pathology of the disease was thoroughly known, and since it has been demonstrated that it is not a sclerotic process the name has been retained. It is usual to discuss otosclerosis under the non-suppurative diseases of the middle ear, but it is as much in place under the non-suppurative diseases of the labyrinth, as in its clinical history it presents forms which closely resemble both of these classes of disease.

Causation.—Practically nothing is known of the causation of otosclerosis, except that in some instances an hereditary influence seems quite marked. One, however, sees patients with this disease in whom the closest questioning fails to obtain any history of the occurrence of deafness in any members of their family, near or remote. It has been found that syphilis and childbirth have a tendency to increase the rapidity of the progress of the disease, but it is not believed that they exert in any way an etiological influence. Several theories have been advanced to account for its occurrence, but they are not much more than the merest speculation. It has been found with slightly greater frequency in females than in males. It may occur at any age. Manasse[1] has

[1] Arch. für Ohrenheilk., vol. xcv, p. 45.

found it in temporal bones of children as young as three years, and it probably occurs earlier in life, perhaps *in utero*.

Pathology.—The essential lesion of otosclerosis is a spongification of the labyrinthine capsule. It is an apparent absorption of the osseous elements and the deposit of new bone of altered structure. This new spongy bone has large Haversian canals, and medullary spaces, with abundant multinuclear cells. These foci may be more or less localized or may be quite extensive, a large part of the labyrinthine capsule being involved; but the line between the diseased area and the normal bone is quite sharply defined. These areas take the hematoxylin and carmine dyes much stronger than the normal parts of the bone. When the process develops near the surface of the bone, it creates an elevation above the level of the surrounding parts; in this way a focus in the niche of the oval window may interfere with the motility of the stapes or, in the internal auditory meatus, may produce a degeneration of the eighth nerve. A favorite location for the process to begin is in the cochlea immediately in front of the oval window. In this location not only by thickening the wall of the niche of the oval window, but by absorption of the annular ligament and the deposit of this new spongy bone in this structure as well as in the foot-plate of the stapes, an ankylosis of the stapedio-vestibular articulation is formed. It may also be noticed that in this location if there is a circumscribed affection of the organ of Corti adjacent to the diseased process in the bone, it would involve that part of this organ which it has been assumed is concerned in analyzing and registering the high-pitched notes of the musical scale. This accounts for lowering of the upper-tone limit, which is not infrequently seen occurring with stapedial ankylosis in this disease. Whether the process begins in the cochlear wall adjacent to the oval window or in some other part of the labyrinthine capsule, there not infrequently results an involvement of the membranous labyrinth which

ends in complete atrophy of Corti's organ. This degeneration of the essential parts of the organ of hearing is characteristic of one form of the disease. The eighth nerve is atrophied and becomes smaller, the spiral ganglion is degenerated and there is more or less complete atrophy of the organ of Corti. There are thus two types of pathological process: In the one, the labyrinthine capsule alone is involved; in the other, in addition to these changes which are essential, there is also involvement of the peripheral part of the perceptive mechanism.

Symptoms.—Deafness is a constant symptom of otosclerosis. If the process in the temporal bone is located in such a place that it does not produce this symptom, neither the patient's nor the surgeon's attention is directed to it. The impairment of hearing may be of any degree of severity. It may develop in an ear previously healthy, or in one the seat of some non-suppurative process. The osteitis metaplastica characteristic of it has been found in the temporal bones of patients who suffered during life with severe deafness and suppurative disease of the middle ear, and this being the case, it follows that it may exist in the residual processes which remain after middle-ear suppuration has exhausted itself. When these diseases exist in the middle ear previous to the development of the otosclerotic changes, it may be difficult to determine when the latter condition develops, as the patient previous to this has deafness from his middle-ear disease. The deafness in otosclerosis may be moderate for some time, or it may become profound or even absolute in a comparatively short time. In this latter instance the perceptive mechanism is involved. Common histories are as follows: That the patient had moderate impairment of hearing for several years, then during from three to six months or so it became very profound; or the deafness may become fairly well marked and remain so for years; or gradually progress until severe deafness ensues and remains stationary, the process never involving the perceptive mechanism. As a rule there

seems to be no difference in the symptoms, except the degree of the deafness and possibly some vertigo, in the patients in whom stapedial ankylosis exists, or those in whom there is degeneration of the perceptive mechanism. The tests, however, are different and will be alluded to under Diagnosis. Many of these patients suffer from tinnitus. This symptom may be the first to attract attention and be much harder to bear than the deafness. It may be continuous or be less marked at intervals. In nervous patients and those who have poor control of themselves it may seriously interfere with the patient's peace of mind, while he can still bear his deafness with resignation. As the deafness becomes profound, the noises may continue or become less marked. In patients with marked deafness with stapedial immotility, the paracusis Willisii is at times present. They hear better in a noisy place (see hyperplastic form of O. M. C. C.). A small percentage of the patients have vertigo. It is not usually severe, but if these patients are asked about this symptom they say they have had "dizzy spells." Rarely the vertigo is severe. In the patients with profound perceptive mechanism involvement, the rule is to find the vestibular function affected. Either the caloric and rotation reactions are absent or very sluggish. Occasionally, however, a patient is seen in whom both of these tests are nearly normal or but slightly reduced. When the lesion is mainly in the conducting mechanism, these reactions are not changed from normal.

Diagnosis.—The diagnosis of otosclerosis is a subject which may not be difficult in some instances, while in others it may be all but impossible. To consider first the disease when the process is located in the inner tympanic wall adjacent to the oval window and produces deafness by interfering with the motility of the stapes but does not produce degeneration of the eighth nerve, the spiral ganglion or organ of Corti. In this type the lesion evidently acts as one of the sound-conduction mechanism. If the deafness is marked, there will be the presence of

Bezold's triad, that is, elevation of the lower-tone limit, bone conduction greater than air conduction, and increased bone conduction. This, however, may occur in O. M. C. C. If there are no indications of disease of the middle ear, the tube patent and the drum membrane normal, one perhaps is inclined to consider the process one of otosclerosis. If, in addition, there is lowering of the upper-tone limit, the process is probably one of otosclerosis. There are two points which would still further strengthen this probability. They are the history of several instances of marked deafness in the patient's family and the "peach-blossom drum membrane." In this characteristic appearance, which was made much of formerly, the membrana tympani is thinner than normal and very translucent, so that the color of the mucous membrane on the internal tympanic wall over the lesion shows through. This mucous membrane is not as pale as normal and therefore produces this pink appearance. It may not be often seen and is in no way necessary for the diagnosis of otosclerosis. If a process as above detailed occurs in a patient who has a chronic catarrhal otitis media, the diagnosis becomes difficult. The tuning-fork reactions may be the same in both diseases. Reliance must then be placed on the family history of the patient, and also lowering of the upper-tone limit. It is claimed by many competent men that the labyrinth may be involved secondary to a non-suppurative otitis media, and that the only indication of this involvement may be a slight lowering of the upper-tone limit. Be this as it may, if the clinical picture of the case is such that the question of the probability of the process being otosclerotic has arisen in the mind of the aurist, the fact that there is lowering of the upper-tone limit is surely evidence of a by no means insignificant nature that the process is one of otosclerosis. In the second type, where the lesion in the bone does not interfere with the motility of the stapes, and there is degeneration of the organ of Corti, the spiral ganglion or the eighth nerve, the tests are characteristic of disease of the perceptive mechanism. If

such a process develops in an ear in which the conductive mechanism is normal, there will be: No elevation of the lower tone limit; B. C.<A. C.; B. C.—; and there may or may not be lowering of the upper tone limit. These patients have profound impairment of hearing as a rule, or even absolute deafness. The evident difficulty is to determine that there is no other non-suppurative disease of the labyrinth which has produced this condition. If this condition occurs in one ear and the opposite one is affected with an otosclerotic process that can be recognized as such, it is safe to infer that the process is of the same nature, if no reason to believe otherwise exists. If the condition is bilateral, one tries to eliminate other diseases which might cause it. The patient's complement-fixation reaction is taken to exclude syphilis. He is questioned as to severe attacks of vertigo which might occur in inflammatory or hemorrhagic conditions of the labyrinth; as to injuries; as to family history of deafness; as to the length of time during which his impairment of hearing was becoming worse, etc. After all of these points are carefully considered and every other probability excluded, one arrives at the conclusion that the patient has otosclerosis affecting the perceptive mechanism. Making a diagnosis by exclusion is not a popular way with aurists, but it has its advantages nevertheless.

In the third class the disease affects first the sound-conducting mechanism and then follows involvement of the perceptive mechanism. If one is fortunate enough, in such a process, to have made tests before the perceptive mechanism has become affected, the nature of the condition may be quite plain. When this is not the case, the tests may show any ratio in the involvement of the two parts of the hearing apparatus, and as no two cases would be exactly alike, to go into detail as to what might be found would only lead to confusion. It may, however, be said that in making the tests in these patients one finds the results such that they can only be accounted for by assuming that there is a lesion in both the sound-conducting and sound-perceiving mechanism.

A great deal of dependence is placed, by some aurists, on the Gellé test in determining stapedial fixation. They believe that if negative it is a strong argument in favor of the process being otosclerosis. It has been shown, under Tests for Hearing, that this simply shows the absence of motility of the ossicular chain, and that if negative it in no way separates a stapedial fixation, the result of the hyperplastic form of O. M. C. C., for instance, from one which occurs in otosclerosis. It is therefore not of much diagnostic value, but it should always be taken and due consideration given to it.

Prognosis.—The prognosis is more or less uncertain. Some patients go for years without getting much worse, while at any time they may become deafer quite rapidly. While recognizing the uncertain nature of the prognosis, the aurist should never state the case thus plainly to the patient, who may be one of those who remain stationary for years, and to point to him the possibilities of his disease would be nothing less than a sin. One has no right to take hope from these patients, neither should he unduly encourage it. The conscientious surgeon will readily avoid either extreme.

Treatment.—If these patients have otosclerosis engrafted upon some catarrhal process within the middle ear, they should be treated exactly as though the former is not present. In not a few patients some amelioration of their deafness arises from a proper and systematic treatment of the middle-ear disease. If the tympanum is normal, no improvement will arise from inflation or any other treatment directed to the middle ear. It may be granted that it is not difficult to determine that the tympanum and tube are apparently normal, but what aurist is there who has not been mistaken on this point. So if there is doubt, as there usually is, the patient should have the benefit of it, and be treated in a very gentle manner as for an O. M. C. C. These remarks apply to the class of cases in which the disease is in the conducting mechanism. When the perceptive mechanism is involved to the exclusion

of the conductive, any treatment directed to the ear is placeboic in nature.

The general health of these patients demand one's consideration. They should be examined by a competent internist and anything wrong corrected. This is a very important item in the treatment of this disease.

As for remedies, very little may be expected. Politzer believes that the disease may at times be kept in abeyance by the internal administration of potassium iodide continued over considerable periods of time, while Siebenmann claims the same results from phosphorus given for months or even years. The patients with perceptive mechanism involvement may be given the iodides and pilocarpine, not with much hope of improvement, however.

CHAPTER VI.

ACUTE INFLAMMATION OF THE MIDDLE EAR.

Anatomy.—A prominent role is played in the acute inflammatory processes of the middle ear by the various folds of mucous membrane existing in the tympanum. These are so disposed that, together with the ossicles and their ligaments, the tympanum is divided more or less completely into two separate parts which inflammatory swelling renders still more complete. The dividing line between the upper part (the vault) and the lower part (the atrium), as seen through the meatus, may be considered as indicated by the short process of the malleus and the anterior and posterior folds. Shrapnell's membrane forming part of the outer wall of the vault is the only boundary of this cavity which is open to inspection, while the membrana tensa affords an indication of the processes taking place in the atrium. The vault is continuous with the mastoid antrum and is in intimate relation with the middle cranial fossa. It will thus be seen that a suppurative process in the vault may extend to the mastoid cells or meninges, while in the atrium it may extend through the oval and round windows to the labyrinth or in rare instances through the floor of the tympanum to the jugular bulb.

Definition.—Acute otitis media is an inflammation of the mucous membrane of the middle ear. As at present conceived it is of an infective nature. It varies greatly in the severity of the reactionary symptoms and in the degree of development and location of the pathological process. To the mild type, in which the inflammation is confined to the lower part of the tympanum and stops

short of the formation of pus and usually of rupture of the drum membrane, the name Otitis Media Catarrhalis Acuta (O. M. C. A.) is given. The severe type in which the reactionary symptoms are usually more intense, and the process affects not only the atrium but in the majority of instances the vault and antrum also, and which, if not aborted by treatment, progresses to the formation of pus, is called Otitis Media Purulenta Acuta (O. M. P. A.). It must be conceded that this division is not all that could be desired and various attempts at a better classification have been made, but so far without success. To call both of these types purulent because they are due to bacterial invasion, no matter what form the reaction may take or how mild the inflammation, is evidently unsatisfactory. This is, however, done by some authors and the term Acute Catarrhal Otitis Media is given to that class above described as Tubotympanites. As has been shown, this is more or less of a passive congestion, and to call it an inflammation is inexact and only leads to confusion. As there are two types of acute otitis media, which may be differentiated clinically and demand different treatment, it seems best to consider them under the heads given above, notwithstanding the fact that the processes are very much alike and differ mainly in their intensity and the degree of their development.

OTITIS MEDIA CATARRHALIS ACUTA (O. M. C. A.).

Acute catarrhal otitis media is an inflammation of the tympanic mucous membrane, limited to the atrium, of comparatively mild type which does not result in the formation of pus.

Etiology.—The disease is very common in children with adenoids and there is no doubt but that these stand in a causative relation. When it results from bathing, it is due to water entering the middle ear through the tube. The habit of diving and after coming up, blowing the nose violently, results in forcing some of the water

which has found its way into the nasopharynx through the tube into the middle ear. The author has noticed that bathing in the more congested locations where bacteria are more abundant often results in the production of the severer form of otitis media. Other causes are taking cold, measles, use of the nasal douche and in general the same as those of O. M. P. A.

Pathology.—The infection probably always gains access to the tympanum through the Eustachian tube. There is an active congestion and swelling of the mucosa extending to the tympanic folds which by their swelling complete the division of the cavity into two separate compartments. The first factor in the production of an inflammation of mild type is this division by which the process is limited to the atrium. If as the serum exudes and mucus is produced, the Eustachian tube is of sufficient patency to afford substantial drainage of these products of inflammation, it constitutes the second factor, while the third is the better resistance of the patient, the fourth being the fact that the infection is of milder nature. As the inflammation subsides, the serum and mucus drain through the Eustachian tube, or are absorbed or are being retained, the process becomes chronic. As the vault does not take part in the process there is no extension to the antrum, the mastoid cells or the meninges.

Symptoms.—The most common symptom is pain. It may be regarded as practically constant. It varies greatly in severity but is usually sufficient to keep the patient awake at night. While the patient also has more or less deafness, perhaps tinnitus, it is not for these symptoms that he seeks advice. It is well, however, to make a record of impairment of hearing which will be found to show the tests characteristic of involvement of the sound-conducting mechanism. In children fever may be present not usually over 103° F., although in infants it may be higher and the attack ushered in by convulsions. Fever is not common in adults although a rise of a degree or so may be met with.

Signs.—The drum membrane presents characteristic changes. The luster is diminished but very rarely, if ever, absent. The color is changed, appearing redder than normal, the degree varying considerably. The membrane may be depressed in the early stages but usually when the patient comes under observation it is normal in position or less depressed than normal. In one type only (to be mentioned later) is its integrity impaired. The change in the structure is not marked, although some thickening and loss of the natural translucency may be present. All of the changes are limited to the membrana tensa. Beyond a physiological reddening of the membrana flaccida no changes are present in this membrane. If the ear is inflated, there will be found to be fluid in the tympanum.

Diagnosis.—That there is an acute inflammatory process in the middle ear is usually readily made out by the pain (and fever in children) with the change in color and luster of the membrana tympani. In differentiating the process from an O. M. P. A. the question resolves itself into whether or not the patient is suffering from a process capable of resolution. If so it is called O. M. C. A. and treated expectantly. In arriving at a conclusion the following points are taken into consideration. (1) Pain: its severity and duration. It is less severe in O. M. C. A. If the patient comes under observation early in the attack, it will still be present and at its height; if later, say on the second or third day, it should be less and diminishing in intensity. (2) Fever: this should be less in O. M. C. A. It is, however, of little value in differential diagnosis. (3) Changes in the membrana tympani. Always limited to the membrana tensa in O. M. C. A. and consisting as a rule of change in color and luster with slight alteration in structure. Bulging or loss of integrity, except as mentioned below, are not present in O. M. C. A. Changes in O. M. P. A. apt to be in Shrapnell's membrane, are discussed under that disease. (4) Finally the occurrence of mastoid involvement excludes O. M. C. A. If upon

examining a patient one feels that he is not justified in postponing incision of the drum membrane, he cannot make a diagnosis of O. M. C. A. The following are three types of history in which the author feels justified in making this diagnosis: (1) A child suffers with pain and fever, usually not over 103° F. This continues a few hours when there is relief from pain and the temperature begins to subside. Soon a seromucous discharge appears, or the mother notices a stain upon the pillow. When the ear is examined twelve to twenty hours later it is found that the drum membrane is healed and in process of resolution or the discharge may continue for a day or so, never becoming purulent. (2) A patient comes complaining of fulness in the head, tinnitus or deafness, and states that from twenty-four to forty-eight hours previously he had severe pain lasting (usually) less than twenty-four hours, when it gradually subsided. The drum membrane is red, the luster is somewhat diminished, bulging is not present, although the natural depression is somewhat lessened and the structure is slightly, if at all, altered. All of these changes will be less the longer the time which has elapsed since the attack of pain. If inflated, fluid is found in the tympanum. This is evidently a resolving O. M. C. A. (3) A patient with the same kind of a process may present himself early, that is, during the height of the pain. In making a decision one takes into consideration that the pain has not lasted long, is perhaps not as severe as one would expect in an O. M. P. A. The drum membrane while somewhat red, possesses some luster, is not bulging, the structure not seriously altered and Shrapnell's is normal or at most shows only a physiological redness. One feels that the process is capable of resolution without incision or rupture of the drum membrane, calls it O. M. C. A., and treats it expectantly. If in twenty-four hours the pain is less severe or absent and the fundus changes no worse or are beginning to improve, the correctness of one's judgment has been demonstrated. If, on the other hand, in twenty-

four hours the pain has not diminished or has increased and the fundus changes have become more marked, one discovers that he underestimated the process and must revise his diagnosis, call it O. M. P. A. and proceed to treat it as such. In comparing the changes in the fundus, reliance must be placed, not so much on the difference in the color, as in the luster, structure and position. With more diminution in luster, increase in the thickness of the membrana tensa and bulging and especially if Shrapnell's membrane shows any sign of involvement, the process declares itself to be a severe one, incapable of resolution without incision of the drum membrane and therefore an O. M. P. A.

Prognosis.—The prognosis of O. M. C. A. is good. The process in the majority of patients ends in cure without much impairment of hearing. Nevertheless, the attack may result in adhesions or thickening of the mucous membrane which produces impaired hearing. This is much more probable to result when repeated attacks occur.

Treatment.—Many attacks may be prevented. The adenoids should be removed. Children with these growths in the nasopharynx are very susceptible to attacks of this form of acute middle-ear inflammation. The mother states that the child is subject to "ear aches" which have probably been attacks of O. M. C. A., and have frequently resulted in more or less impairment of hearing when the patient comes under observation. Removal of the adenoids usually results in the relief from future attacks. The tonsils if enlarged should also be removed, although their influence in producing this condition is much less. Bathers should be cautioned against diving and then forcibly blowing the nose, and congested pools or parts of the shore should be avoided. The nasal douche should be discarded and an atomizer substituted if it is necessary to cleanse the nose.

In treating the attacks no ear drops are to be used. They do no good. A patient who gets well after the

instillation of any of the various compounds devised and prescribed either by the laity or others has recovered in spite of, but not on account of, them. While being of no use they mask the appearance of the fundus changes so that one is often in doubt as to the treatment to pursue. A very gentle inflation sometimes helps these patients. The nose and tubal orifice is anesthetized with a solution of cocaine before inflation. One or two quite gentle compressions of the bulb should be made, just sufficient to force air into the tympanum. No attempt to drive the secretion from the tympanum through the tube, such as is made in tubotympanic congestion, is justifiable. After inflation a solution of silver nitrate, 4 per cent., is applied to the mouth of the tube. If this treatment results in improvement, it may be repeated in twenty-four to forty-eight hours. Or if one feels timid about inflating such a patient, the silver nitrate solution may be applied, after anesthetizing the tubal orifice and the inflation dispensed with. Heat applied to the side of the head and ear often seems to relieve the pain and make the patient more comfortable. Anodynes, such as any of the preparations of opium, should not be administered, as they mask the pain upon which a decision as to the necessity of a myringotomy may eventually rest. After the acute symptoms have subsided the patient should be inflated and the silver solution applied to the tube every two to three days until the tympanum is dry and the hearing becomes as near normal as can be expected.

OTITIS MEDIA PURULENTA ACUTA (O. M. P. A.).

Acute purulent otitis media is the severer form of inflammation of the mucous membrane of the tympanum, which if not aborted by myringotomy (as it only occasionally can be) results in the production of pus.

Etiology.—The disease may follow a cold in the head, especially if the accessory sinuses are involved. Chronic sinusitis is also frequently a cause. The purulent secre-

tion from these cavities finds its way into the nasopharynx, then by blowing the nose, it is forced into the middle ear through the tube. If the epithelium covering the tubal mucosa is absent through inflammation or any other cause, the protection against bacterial invasion afforded by the cilia is lost. This cause, doubtless, operates in many of the nasopharyngeal infections to make the tympanum more vulnerable. Adenoids furnish a favorable field for the lodgement and development of bacteria and also interfere with the proper aëration of the middle-ear spaces and are a frequent cause of the disease. The use of sprays or nasal douches when adenoids are present often leads to forcing infection through the tube. Enlarged tonsils may be a factor in the causation but not as frequently as adenoids. Atrophic rhinitis, by furnishing a constant supply of germs in the nose and nasopharynx near the tubal orifice, is an ever-present menace. These patients are frequently the subjects of middle-ear inflammation. Indeed it seems strange that so many of them escape. When they seek advice and a nasal douche is prescribed to remove the crusts, the conditions favoring middle-ear infection become almost ideal. Nasal and nasopharyngeal operations are not infrequently responsible for an O. M. P. A. If it becomes necessary to use a postnasal tampon, the danger is increased. For this reason this device should be avoided in epistaxis if it is at all possible to control the hemorrhage otherwise. Tuberculosis frequently causes the disease (see Tuberculosis of the Ear). Scarlatina, diphtheria and measles have lesions of the nose or nasopharynx which may extend to the ear and in fact frequently do so. The disease occurs in pneumonia, typhoid fever, influenza, variola, in fact any of the infectious diseases. Injuries of the membrana tympani, especially with infected instruments or foreign bodies, are at times responsible for O. M. P. A.

Pathology.—The atrium may be involved alone but this is the exception. The vault may be involved alone, the infection passing through the atrium and gaining

access to the vault, which becoming walled off by the swelling of the mucous membrane folds, undergoes a suppurative inflammation, while the process in the atrium, never being severer than an O. M. C. A., subsides, leaving the disease localized in the vault. Or both divisions of the tympanum may be involved at the same time, which is the usual condition. Postmortem examinations have shown the tympanic mucous membrane to be swollen and infiltrated with a serous exudation and presenting ecchymotic areas, the epithelium, cloudy, swollen and cast off in places. The infection usually reaches the tympanum through the tube, occasionally through a solution in continuity of the drum membrane. That it is possible for the infection to be carried to the tympanum through the blood stream or lymph channels cannot be denied, but the instances in which it does so are rare. Many different bacteria are capable of producing the disease. The more common reports from the laboratory on the results of the examinations of smears or cultures of the aural pus are: long- or short-chained streptococci, pneumococci, Streptococcus capsulatus, staphylococci or mixed infection. Occasionally some of the rarer forms are found.

Symptoms.—Pain is a predominant symptom of this disease. It may be located in the ear or radiate over the side of the head. While there may be remissions, it is usually more or less constant until the drum membrane has either been incised or ruptures when it usually subsides. It is apt to be worse at night and in the recumbent posture. The patient usually describes it as "boring or throbbing" in character. Very rarely the patient may state that at no time has he had any pain. This frequently occurs in tubercular process but outside of these is seen occasionally. As the pain is caused by tension either from swelling of the mucous membrane or the accumulation of secretion, it seems strange that sufficient tension to produce rupture of a previously normal drum membrane can exist without pain, but it does so, although

very rarely. Fever frequently occurs in children. In infants the temperature may reach 105° F., and the attack begin with convulsions. In older children the temperature seldom rises above 103° F. or 104° F. from the otitis media alone. Adults may have some fever but it is not uncommon for the disease to be afebrile. With a temperature of 103° or even less in an adult or adolescent one suspects the presence of some complication. Tinnitus may be an early symptom before the disease is sufficiently developed to produce pain and it is frequently complained of as the process is subsiding. During the height of the process it does not usually attract the patient's attention on account of the pain and discharge. The same is also true of deafness which is invariably present to a greater or less degree. As the reactionary symptoms subside the impairment of hearing is very frequently complained of. It arises from lack of motility of the drum head, from thickening of the mucous membrane which interferes with the vibrations of this structure or those in the oval and round windows, or from fluid accumulating in the region of the fenestræ. Except the labyrinth is involved the deafness shows the tests characteristic of disease of the conducting mechanism. Vertigo if present should always lead one to suspect labyrinthine disease, although it may occasionally be due to pressure upon the fenestræ or to vascular disturbance arising from the severe inflammation in proximity to these windows. In about 10 per cent. of the patients there develops loss of one or more of the sensations of taste on the anterior two-thirds of the tongue on the involved side. Either the ability to perceive all four of the primary tastes, acid, bitter, sweet and salty, is lost, in which event the corda tympani is totally involved or some of them may be perceived when it may be presumed that the corda is but partially involved.

Discharge is a constant symptom. If it follows an early incision of the drum membrane, it is usually serous or serosanguinolent for a day or so, but if it persists it

becomes purulent. If spontaneous rupture of the membrane occurs early, the discharge may have the same appearance for a day or so, soon to become purulent. When the rupture takes place later or the incision has not been made early, pus usually is the major content of the discharge after the blood is washed away. Outside of these considerations the amount and character of the discharge vary greatly. It usually continues free until the height of the process is passed, when it is apt to become less purulent and more mucoid in character and gradually cease. At times a free myringotomy seems to abort the process and the discharge never becomes frankly purulent but is serosanguinolent and continues a few days only, containing, however, pus cells and bacteria in abundance. In other patients the discharge becomes very free and leads one to suspect the presence of a mastoid abscess; being much more in amount than could come from the middle ear. Between these two extremes any variation may occur. The otorrhea instead of diminishing to a cure in from one to six weeks may persist, passing into a chronic condition. This is the usual occurrence in tubercular patients and is frequent in children with adenoids and when the disease follows scarlatina and diphtheria. In infants with marked patency of the Eustachian tube sufficient relief to the tension in the middle ear may be afforded by discharge through this passage, so that the membrana tympani does not rupture. This process if unrecognized may continue for some time, the child having a varying amount of pain and fever.

Signs.—The color of the membrana tympani is changed. In the beginning of the process the membrane is injected along the manubrium and the tympanic ring, soon the change in color extends to the membrana tensa which often becomes a bright red. As the process goes on the color usually diminishes to a dull or even livid redness. There may be ecchymotic spots or hemorrhagic bullæ, especially in the influenzal otitis media. If there are

interstitial abscesses or the membrane becomes thin from the pressure of the pus and is about to break down, yellowish areas may at times be seen, which indicate where rupture is about to take place. The luster of the drum membrane is due to the peculiar character of its epithelial covering, and for the luster to be normal this layer must be intact. As this is affected by the inflammation, the luster diminishes to a varying extent, depending upon whether the epithelium is merely changed by the exudation or exfoliates *en masse*. To a certain extent color and luster are correlative. The brighter the color, the less the loss of luster, as in the early stages; while in a type which is sometimes seen a little later in the disease the loss of luster is absolute, the membrane appearing not unlike wet blotting paper. This type of membrane is due to death, *en masse*, of the epithelial covering and shows no redness whatever, although if the macerated epidermis is removed, as it usually can be, the membrane appears red. Care must be taken not to mistake for luster the light reflex from secretion which may cover the drum membrane. Sometimes these reflexes are pulsating in appearance due to the effect of the cardiac contractions on the intratympanic blood supply. In exceptional instances the membrana tensa may be nearly normal in color and luster. In these the process involves the vault alone, the disease in the atrium having resolved and the changes are in Shrapnell's membrane. This structure frequently appears altered in color, showing varying degrees of redness, but the color changes in Shrapnell's are as a rule not of as much importance as changes in position. Theoretically, the drum membrane should be depressed from tubal occlusion at the very beginning of the attack. Patients are practically never seen at this stage. When they come under observation, the position of the membrane is either less depressed than normal or bulging. This outward movement of the drum head is due either to the swelling of the mucous membrane internal to it, or to the accumulation of

secretions. It may involve the whole membrane, Shrapnell's included, or be limited to the membrana tensa or some part thereof. When the whole membrane bulges, the manubrium may lie in a gutter where it holds the membrane relatively depressed. If the manubrium can be made out, it often seems lengthened, the converse of the foreshortening with depressed membranes. If the bulging is localized, it may be in the posterosuperior quadrant and extend into Shrapnell's membrane. These bulging areas usually collapse after rupture, but before this occurs a conical process may be produced which remains after a small perforation has occurred. These conical perforations are often associated with mastoid abscess. When occurring in Shrapnell's membrane, they may attain considerable length and in extreme instances even present at the external end of the meatus, very closely resembling an aural polyp.

When the patient comes under observation rupture may not have taken place, the membrane being intact, or there may be a perforation. Usually there is but one. In tubercular process there may be more than one. Spontaneous perforations are perhaps more frequently found in the anterior half of the drum head. If difficulty is experienced in locating the perforation, the expedient suggested under "Examination of the Patient" should be tried. Inflation may thus show the location of the opening and give valuable information as to its size and the sufficiency of drainage. As the process resolves the perforation usually heals with cicatricial tissue before the changes in the intratympanic membrane have completely subsided. At times a discharge occurs without a perforation in the drum head, the pus dissecting its way out through the Rivinian segment beneath the periosteum, and producing a rupture on the canal wall. In infants in exceptional instances the drum head may remain intact, the pus escaping through the Eustachian tube. The structure of the membrane is altered by infiltration with inflammatory products and swelling of its mucous and

fibrous layers. In interpreting this sign, one notices that the membrane, independent of its loss of luster, seems less translucent than normal. When the thickening is extreme, it may be impossible to make out the manubrium, the short process is obscured and the drum head seems opaque. Change in structure is regularly present in O. M. P. A., and in membranes without marked diminution in luster or change in color it becomes a valuable sign in diagnosis. The tough, thickened membranes which fail to rupture spontaneously are among the most dangerous, in view of the complications which they produce by delaying the establishment of drainage.

Diagnosis.—That the patient has an acute middle-ear inflammation is readily determined by the symptoms and history together with the otoscopic examination. Whether the process is an O. M. C. A. and may be treated expectantly, or is an O. M. P. A., and demands myringotomy, has to a certain extent been discussed under the former disease. It may be added, however, that the diagnosis of O. M. P. A., and therefore the indication for myringotomy, rests on the following points: severe pain, especially if associated with mastoid tenderness; fever attributable to the aural process; bulging of the drum membrane, especially if in Shrapnell's or conical in form; a yellow area in the membrane indicating that pus is about to break through; marked diminution of luster, especially if the membrane resembles wet blotting paper; marked change in structure. Any one of the drum membrane changes given above, if associated with pain or fever, indicate the necessity of myringotomy. If the pain is mild or even absent, the fundus changes alone enable one to decide. After the membrane ruptures and the discharge appears, the diagnosis is established by this symptom alone. If an otitis externa has produced occlusion of the meatus or the same result has been caused by strictures or exostoses, so that a view of the fundus cannot be obtained, the diagnosis before the appearance of the discharge may be very difficult. One would then

rely on the intensity of the pain, the amount of fever, and perhaps also the occurrence of mastoid tenderness. After the discharge appeared its quantity, nature, and bacterial content would enable one to decide.

Prognosis.—The result of an acute purulent otitis media is always more or less doubtful. A great many patients recover and have very good or even slightly diminished hearing. Others recover with more or less deafness or tinnitus. These symptoms are due more to the resulting intratympanic changes than to any effect produced upon the drum head. Adhesions between the ossicular chain and tympanic wall, as well as thickening of the mucous membrane in the niches of the labyrinthine fenestræ may remain and profoundly affect the hearing. One sees at times ears which are totally deaf as the result of a suppurative process. Upon testing them loss of hearing is found to be due to a lesion of the perceptive mechanism, doubtless due to a labyrinthitis which occurred at the time of the attack. The perforations in the membrane usually heal with more or less cicatricial tissue, according to the amount of sloughing in the original process. Some cases become chronic no matter how carefully treated. Mastoiditis of mild degree occurs in every patient who has involvement of the vault, and this is in the majority. It may resolve or demand operation. Sinus thrombosis, meningitis, brain abscess or labyrinthitis are other complications which are far from rare. No one is able to tell in advance the patients who will develop these complications. So the prognosis, while guarded, should be encouraging. Bacteriological examination of the aural pus may throw some light on the probable termination, although too much reliance should not be placed upon it. The Streptococcus capsulatus and to a lesser degree the pneumococcus are the cause of processes which show a tendency to invade the mastoid and produce mastoid abscess and other complications (see Mastoiditis). These infections influence the prognosis in that the progress of the disease is more insidious and less

painful and therefore its extent may not be realized. The prognosis is not so good in diabetics and others with low resistance. The processes caused by scarlatina and diphtheria are more apt to become chronic than other types, except the tubercular, which is almost certain to do so.

Treatment.—When a patient suffering from this disease presents before spontaneous rupture of the drum membrane has taken place, myringotomy is indicated. As long as it is safe to hope for resolution, one considers the process an O. M. C. A., and treats it accordingly. But once it is seen that this is not probable to occur, the diagnosis is changed to O. M. P. A. and the membrane is incised. In coming to this decision the benefit of any doubt should be cast upon the side of a prompt incision of the membrane. The process is one in which the complications are serious and the best that can be done to avoid them is to perform an early and free myringotomy. Then the resulting scar is less if this is done early before necrosis of an area in the drum head results, or spontaneous rupture occurs. An incision heals with a linear scar, while if rupture occurs a greater or less area filled in with cicatricial membrane may be expected. Incision is also the best means to adopt for the relief of pain. It is a bad plan to give these patients anodynes, as the pain is an important indication as to the progress of the process. They certainly should not be given before incision and as this operation usually relieves the pain, unless there are complications, it is unwise at any time to mask this symptom which may afford so much information. In children the temperature may not fall for a day or two after an incision has been made. If one feels certain that the atrium alone is involved, the incision may be limited to the membrana tensa, but if the vault also is taking part in the process it should extend through the posterior fold into Shrapnell's membrane. When the patient is seen after spontaneous rupture of the membrana tympani, it remains to determine whether or not sufficient drainage has been established. If the pain is

relieved and fever is absent, especially if the perforation is of fair size, one is justified in omitting to perform a myringotomy. On the other hand, if pain or fever are present, especially if the perforation is a small one, the drainage is presumably insufficient and an incision should be made. Having provided for drainage, the ear is syringed every two to four hours, according to the amount of discharge. A solution of bichloride, 1 to 5000, or boracic acid, one dram to the pint, may be used. These irrigations should be as warm as can be borne with comfort. Eight ounces of fluid if properly used will be suffi-

Fig. 48.—Soft-rubber bulb syringe.

cient to cleanse the ear. Just sufficient force to remove the secretions should be used. A soft-rubber bulb is a very safe syringe for these patients (Fig. 48). The ear should be drawn upward and backward, or if the patient is an infant or young child downward and backward and the tip of the syringe placed behind the tragus, but not allowed to enter the canal, and the current directed in the line of the axis of the meatus. After syringing the ear the meatus is dried. The wearing of a wad of cotton in the ear, while it may seem necessary in exceptional instances, is not to be advocated as a routine. The question of whether or not to perform a second myringot-

omy in a given patient may arise. This will probably occur if relief from the symptoms has not followed the first one, or if after the symptoms have become better, they recur coincident with the closure or diminution in size of the perforation. Before determining to make a second incision of the drum membrane, if it is known that the first one was properly made, it is well to investigate the condition of the mastoid as it will usually be found that a mastoid operation is needed and not a myringotomy. Some surgeons go so far as to say that a second incision should never be made, but a mastoid operation instead. If a free incision does not relieve the symptoms, the inference is justifiable that the obstruction to drainage is not in the membrana tympani, but is more deeply seated and that a second myringotomy would be useless. If the symptoms (pain, fever or mastoid tenderness) are relieved but recur with closure or contraction of the perforation, and the process is still in the first week dating from the inception of the pain, a second myringotomy is justifiable. The author has in mind a patient upon whom he performed the second incision on both sides, under these circumstances, and although the infection was the Streptococcus capsulatus, the patient made a good recovery without complications and had remained perfectly well when seen three years later. Another question which may arise is, When should the adenoids be removed? Some surgeons of great experience in treating the aural diseases of children, remove the adenoids at the same time that the myringotomy is done, claiming thereby to lessen the duration of the suppurative process and to minimize the danger of complications. This practice has, however, not become general. It is perhaps the rule, that if the process is not resolving as shown (mainly) by the lessening of the discharge in the second or third week, to remove the adenoids, unless it is probable that the continuance of the discharge is due to mastoiditis. However, each case must be decided by itself, but in no instance should the adenoids be allowed to remain until the dis-

charge becomes chronic. The general health and nutrition of the patient recovering from an acute suppurative process of the middle ear should also receive attention, and appropriate measures adopted.

After the inflammation has run its course and the drum membrane is healed, an attempt is made to limit the amount of resulting deafness. Inflations, by breaking up adhesions and producing a pneumomassage of the mucous membrane, may be of value. They should not be begun until the drum membrane is in process of resolution, and the luster and structure are returning to normal. Inflations must be very gentle at first until it is ascertained that they do not produce painful reaction. They are to be followed by the application of nitrate of silver or argyrol to the mouth of the tube in the same manner as in tubotympanites, and continued at intervals of two or three days until the ear becomes dry, the tube remains patent and as much improvement of hearing gained as can be expected. It should, however, be ascertained that the impairment of hearing is due to lesion of the sound-conducting mechanism before persevering in this treatment.

MYRINGOTOMY.

Instruments.—The proper instrument for incising the drum membrane is a small scalpel (Fig. 49). The cutting edge may be convex, straight or slightly concave, accord- to the preference of the operator. Lance-pointed knives

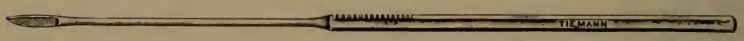

FIG. 49.—Myringotomy knife.

should be avoided as the intention is to incise not to "pierce" the membrane.

Asepsis.—The operation should be done in a thoroughly aseptic manner. If the normal membrane is incised in

this way, no reaction will take place, but the margins of the incision will heal in a few days, at the longest. At times the operation may be done when infection is absent from the middle ear, and in any event, whether this is the case or not, infection should not be introduced by the surgeon. The ear is to be syringed with a solution of bichloride of mercury, 1 to 2000, dried, and the meatus wiped with bichloride and alcohol. The auricle and surrounding parts washed with soap and water, dried, then wiped with gauze saturated with alcohol and bichloride. The hands of the surgeon should be thoroughly cleansed and all instruments sterilized.

Anesthesia.—General anesthesia is preferred. Nitrous oxide gas, being rapidly administered and recovered from without unpleasant effects, is the anesthetic of election. When it is not available and the patient is a child, a few whiffs of chloroform, just sufficient to produce primary anesthesia, is all that can be desired. Local anesthesia, although used by many, has not become popular. Advocates of this method use a mixture of menthol, carbolic acid and cocaine, equal parts. They should be rubbed together in a mortar without the addition of even a drop of alcohol or other fluid. The drum membrane is painted with this mixture in the line in which it is proposed to make the incision. As the parts become blanched, anesthesia is usually as profound as it will become, and the incision is made. The objections to this method are that it does not always cause a painless operation, and more or less reaction of the membrane and walls of the canal takes place. This inflammation and swelling interferes with the proper appreciation of the subsequent progress of the disease. While anesthesia is always desirable, it is possible to perform the operation without it.

Incision without Anesthesia.—If this is attempted, the head of the patient must be firmly held by an assistant so that the patient will not move away from the operator as the knife touches the drum membrane. The patient's hands should also be held so that he does not

grasp the surgeon's arm as the incision is being made. The knife is held by the index finger and thumb of the right hand in such a manner that when introduced into the canal and the point is near the drum head, the other three fingers rest upon the mastoid or cheek, according to whether the left or right membrane is about to be incised (Fig. 50). The thumb and finger thus holding the knife are capable of extension, while the other three fingers maintain their rest upon the patient's mastoid or cheek, and thus the whole hand moves with the patient's

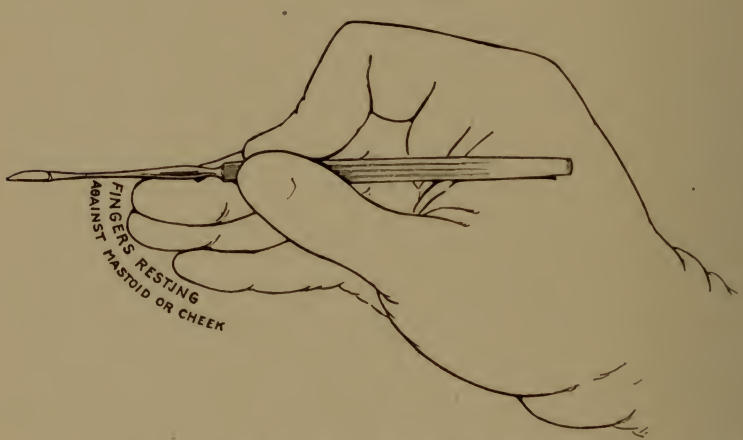

Fig. 50.—Position of the hand and fingers while holding the knife in incising the drum membrane. The thumb and index finger extend and complete the incision.

head while the incision is being made by the excursion of the thumb and index finger. Thus one is able to incise the membrane, notwithstanding a moderate moving away of the head. This method of holding the knife has another advantage in that the incision is made by movement of the thumb and finger, which is much more delicate than it would be if executed with the whole arm or hand. If, therefore, the blade of the knife encounters the long process of the incus, it is more readily turned aside. It will also be found very convenient even if the

patient is anesthetized, although in this case any method of holding the knife that appeals to the operator is permissible as the incision is performed in a more deliberate manner.

Location of Incision.—The incision is usually made in the posterior part of the drum membrane. Circumstances are very rare which render it desirable or advisable to make the incision anterior to the manubrium. The posterior half is more accessible and if it becomes necessary to extend the incision into Shrapnell's membrane, as is frequently the case, it can readily be done from the posterior position, while if the incision is made anterior to the manubrium, it would be done with difficulty. Moreover, the part of the tympanic cavity which it is desired to drain is the posterior which is adjacent to the posterior part of the vault communicating with the antrum.

Anatomical.—It is necessary to bear in mind the anatomical structures on the internal tympanic wall; the niche of the round window, the oval window containing the stapes articulated with the long process of the incus, and between these two fenestræ and anterior to them, the promontory which approaches very near to the tympanic membrane when the latter occupies its normal position. When it is necessary to incise the drum membrane, it has frequently, on account of the bulging, receded from the promontory so that the depth of the cavity has increased.

Making the Incision.—The incision should always be made in an upward direction, not only because it is more convenient, but because it is safer also. The jugular bulb is in relation with the floor of the tympanic cavity. At times the bony structure which covers it in this location is very thin or absent, so that the knife, if directed downward, may wound the bulb. In rare instances the dome of the jugular bulb rises into the tympanic cavity, when it would almost certainly be wounded if the incision was made downward and might be, no matter how the incision was made. Fortunately this anomaly is rare and the

possibility of its occurrence should not make the surgeon timid in performing this operation. The knife is entered as low as possible and the incision made upward, parallel with the tympanic ring (Fig. 51). If it is desired merely to open the atrium, the incision stops just short of the posterior fold. This may be the case in mild processes limited to the atrium; usually, however, it is best to continue the incision through Shrapnell's membrane until the knife impinges upon the bone in the Rivinian segment. It is then drawn out along the posterosuperior wall for about one-fourth of an inch, cutting the soft parts down to the bone. This part of the incision lies directly over the antrum and is supposed to deplete the tissues and in

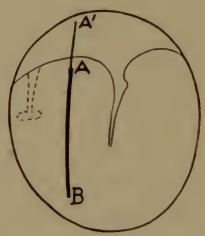

FIG. 51.—Incision of the membrana tympani. *A–B*, incision totally in the membrana tensa. *A'–B*, incision extended through Shrapnell's membrane.

some way produce a beneficial effect on an inflammatory process within the antrum. When the point of the knife has penetrated the membrana tympani, it usually comes in contact with the promontory. If it does not, no attempt should be made to extend it until bone is encountered. The point of the knife may be directed toward the oval window or the niche of the round window, and if the surgeon believes that he must pass the knife until its point encounters bone, the labyrinth may be penetrated with disastrous consequences. As the blade of the knife moves upward, the long process of the incus may be encountered. The instrument should be held with a light grasp and allowed to deviate to avoid injury to this structure.

Sufficiency of the Incision.—After the incision is made, as the operation is so soon completed, the beginner frequently feels that not enough has been done. This desire to go in again and "complete the work" should be restricted, as nothing of value can be accomplished in a field so obscured by hemorrhage. For the same reason, it is impossible by inspection to ascertain whether or not the incision is sufficient. Inflation might decide this question, but it is usually a bad practice to inflate an ear which has just been incised. It will be much easier to determine this on the following day both by inspection of the drum membrane and the amelioration of the symptoms, which will usually take place if the operation is properly performed. The discharge may also furnish some information on the subject.

Accidents.—The amount of bleeding after the operation varies greatly. At times blood will only appear externally at some interval after the incision has been made, while, again, the external auditory canal may become at once filled with blood. If the jugular bulb was wounded, this would occur before the operation was completed. For the ordinary hemorrhage nothing should be done, as the depletion caused by it is in every way desirable. If, however, it is so free or persistent that injury to the bulb may reasonably be inferred, it must be checked. This can usually be done by packing the canal firmly with gauze and an outside pad applied in such a manner that pressure is exerted upon the gauze within the meatus, the whole to be held in place by a snug bandage. The patient is put to bed and observed and treated in the manner outlined under Sinus Thrombosis. There is naturally danger of this condition developing, but it may not occur.

In the operation as described above there is no danger of the carotid artery being wounded. If the labyrinth is entered through the oval window (or through the round window, although this possibility is denied by many), labyrinthitis is a natural sequence. The best way

to avoid this accident is to familiarize oneself with the exact location of these structures and to avoid them when entering the knife, and also by brilliant illumination of the field and the use of as large a speculum as possible, to secure such a view of the drum membrane that one appreciates the moment the point of the knife passes through this structure, independent of the sensation communicated through the knife to the fingers. Another accident which fortunately but rarely occurs is facial paralysis, the nerve being injured by the knife as it sweeps upward. The writer has known of this in but one instance. The operation was performed by one of his assistants, who was a capable man. This accident may be avoided by not allowing the point of the knife to follow along the internal tympanic wall. The chorda tympani is not often wounded in the operation. The point of the knife usually passes clear of the nerve in the upward sweep. Ten consecutive patients tested for change in taste sense showed the same reactions after as before the operation.

CHAPTER VII.

MASTOIDITIS.

ANATOMY.

The mastoid portion of the temporal bone lies behind the external auditory canal, extending above to the squamous portion and posteriorly to the occipital bone. It may be regarded as a truncated wedge, the base being subcutaneous and the apex joining the petrous portion. It is in relation: above, with the middle cranial fossa; behind, with the posterior fossa; the posterior meatal wall forms its anterior surface; while below, it is grooved for the attachment of the posterior belly of the digastric muscle. Projecting downward from the outer part of the inferior surface is the tip of the mastoid, which is a conical process giving attachment to the sternomastoid, trachelomastoid and splenius capiti muscles. At the antero-internal border of the truncated wedge is the descending limb of the Fallopian canal containing the facial nerve.

Variations.—The mastoid outlines are subject to many variations in position. Thus, the middle fossa may be very low and the posterior well forward, in which event there will be a small mastoid, or the converse may be true when a large mastoid is the result. In these large mastoids the tip is usually of greater size and they are, as a rule, of the cellular type; while in the small ones the bone is apt to be of more dense formation. It seems probable that in the development of the bone the cells form from the antrum into the mastoid, thus forcing back and arresting the encroachment of the middle and posterior fossæ, producing the cellular type of the large

mastoid. Why, in one bone this process goes on until a large roomy mastoid is the result, while in another but few cells are formed and the bone remains to a large degree in its infantile type, it is impossible to say. The bone at birth contains the antrum and a few cells lying adjacent to it. Throughout the rest of the mastoid process the structure is composed mainly of diploetic tissue which is separated more or less completely from the antrum by a denser lamina of bone. There is even

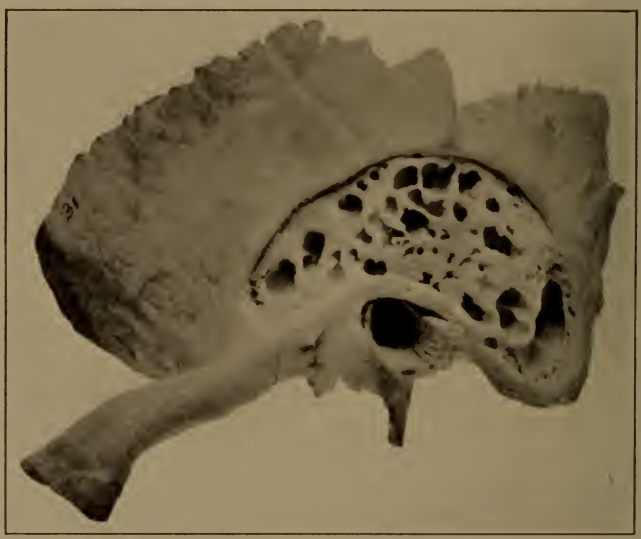

Fig. 52.—Pneumatic mastoid, cortex removed. External view.

at this early stage a great variation in the amount of diploetic tissue, and therefore of the position of the sinus. In a mastoiditis in an infant this tissue may be completely broken down, laying bare the sinus plate or even the sinus, or it may have resisted invasion. It is in this diploetic tissue that the cells of the mastoid are formed and even at this early stage of development of the bone its future type may be foreshadowed. According to this variation of the contents of the mastoid process, the bones

are usually divided into pneumatic, diploetic and sclerotic types. These types can be recognized more or less plainly at operation or upon examining dry specimens and have without doubt a great influence in deciding the form which an inflammatory process in the mastoid may take.

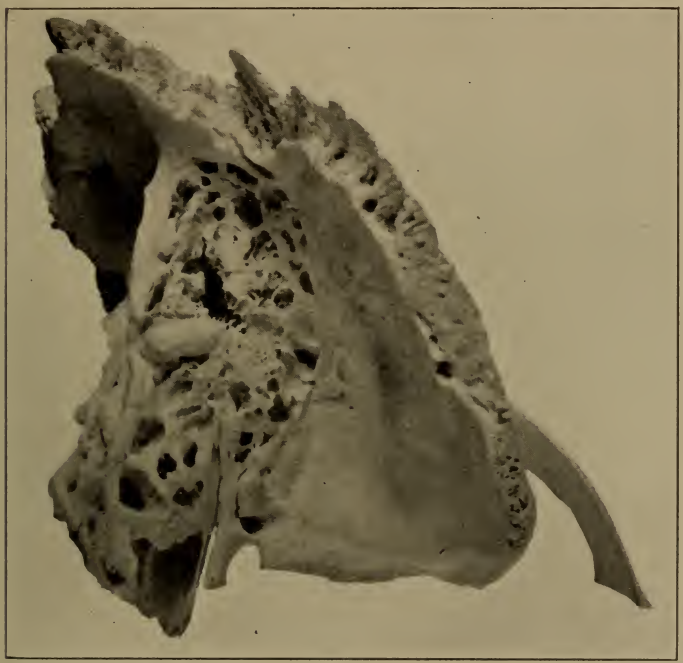

FIG. 53.—Pneumatic mastoid. Superior view of temporal bone. (Mr. Burchell's dissection.) The inner table removed showing the cells extending well into the petrous tip.

Pneumatic Type.—In the fully developed pneumatic type of bone, the mastoid cavity is completely occupied by cells, the septa between which are thin and shell-like (Figs. 52, 53, and 54). They extend into the tip, frequently back over the sinus, at times even into the occipital bone; into the angle formed by the middle fossa and the recession of the sinus plate above the knee, forward

into the posterior root of the zygoma and upward into the squama. This is a distinctly pneumatic type. On the other hand, one sees bones with a thick ivory-like cortex and a few cells with thick sclerotic walls, really a sclerotic mastoid containing a few cells. Between these two extremes may be found any grade in the degree of the cellular development of the bone. When but a few cells are present, it can readily be seen what an

FIG. 54.—Pneumatic mastoid, showing the interior of the mastoid process and the tympanum.

influence their location will have on the direction which a progressive infective process will take. If they are deeply placed and extend from the antrum to the inferior surface of the bone, a purulent accumulation will find its way through this avenue and appear beneath the sternomastoid in the neck (Bezold type). If the cells extend toward the zygoma, the pus will appear in this locality as a subperiosteal abscess. These are but two instances of their effect upon the process, although there are many others. It

is in these hard bones but slightly pneumatic that one is more apt to encounter atypical cases of mastoiditis.

Diploetic Type.—In the diploetic type the cortex is usually thicker and the mastoid and tip smaller, and in place of cells the mastoid is occupied by osseous tissue, very much resembling the diploe between the tables of the calvarium. As a rule there are a few small cells

FIG. 55.—Diploetic mastoid with pneumatic spaces near the antrum.

about the antrum between which and the surface the bone is hard, and below this extending into the tip the diploetic tissue is found (Fig. 55). Or the bone in the neighborhood of the antrum may be pneumatic with a diploetic tip.

Sclerotic Type.—In the sclerotic type (Fig. 56) the bone is hard and ivory-like, but never as dense as the labyrin-

thine capsule. The antrum varies in size and may be the only cell in the mastoid. If the antrum is large, this is usually the case; if small, it may be so or there may be a few small cells adjacent to it. These bones are very apt to have diploetic tips. Two theories are advanced to explain the development of this type of mastoid. One is that the condition is caused by suppuration resulting

Fig. 56.—Sclerotic mastoid with large antrum.

in the formation of osteoblastic granulations in the cells of a pneumatic bone, which later form bone, obliterating the cells and resulting in a sclerotic mastoid. This process has been observed by Dixon at the New York Eye and Ear Infirmary by means of *x*-ray plates. A series of exposures have been made in several patients and the different phases of the process shown. In these observations the development of the sclerotic bone followed

upon the resolution of an acute mastoid inflammation. All operators of experience have opened mastoids in which the cells throughout contained these large osteoblastic granulations, the pus being only found when the antrum was opened. Such a mastoid is taking care of itself in a thoroughly efficient manner and may be capable of spontaneous cure, although the suppurative process in the middle ear may pass into the chronic stage. As the sclerotic bone is more often encountered when operating for chronic suppurative processes, it has been assumed that it is the result of chronic suppuration. The observations of Dr. Dixon tend to show that it is in the acute stage and while the condition is becoming chronic, that the change takes place; although it is a well-established fact that new bone formation occurs in chronic suppuration, either in the form of osseous processes projecting into or obliterating the antrum, or as exostoses in the tympanic cavity, or on the surface of the mastoid. The other theory, advanced by Cheatle, of London, is that these are infantile types of bone, that have failed to develop the pneumatic spaces. Probably both theories are true, as one meets with two types of sclerotic mastoid; one with low middle fossa, forward sinus groove and diploetic tip—a small bone which failed to develop—the infantile type; the other, the middle fossa is not low, the sinus not forward and the tip is sclerotic—this is not a small bone but has been sclerosed as a result of a suppurative process.

Mastoid Antrum.—The mastoid antrum is a small cavity which may be regarded as practically constant, situated at the junction of the mastoid and petrous portions of the temporal bone. It communicates with the vault of the tympanum by the additus ad antrum, which is a name given to the junction of these two cavities. The additus must not be considered as a passage, as it is not, but merely a name given to a certain part of one cavity which is formed by the tympanic vault and the mastoid antrum. In small antra the additus does not represent a constric-

tion between the vault and antrum. In large antra which extend well into the mastoid, a condition not infrequently found at radical operations, it still must not be regarded as a constriction, although the antrum narrows to a considerable degree before it joins the tympanic vault. Certain pathological processes, of infrequent occurrence, can only be accounted for upon the assumption that the additus may be obstructed by swelling of the mucous membrane or granulations, so that the antrum and mastoid become completely separated from the vault. The antrum is in relation with the middle cranial fossa above separated by a thin shell of bone, the tegmen antri. The horizontal semicircular canal forms part of the floor of the antrum anteriorly, and lying upon this canal in the region of the additus is the short process of the incus.

Landmarks.—When it becomes necessary to open the antrum it may be located in the following manner: first draw a line tangent to the upper canal wall, then one tangent to the posterior wall. These imaginary lines map out a triangle (the Dench triangle) of which they form two sides, the base being formed by the wall of the meatus. In this triangle at a depth of about one-half inch, lies the antrum. In this triangle there is often found a small spike-like process, the spine of Henle. This is taken by some operators as a guide to the antrum; but as it often projects from the extreme margin of the entrance to the osseous meatus, and as it is far from constant, it is less reliable as a guide than the triangle as described. Another method of laying out a triangle is that of MacEwen. The lower temporal ridge forms its upper limit, the posterior margin of the meatus, its anterior boundary, while the third side is an imaginary line connecting these two. It is not an infrequent occurrence to find either the sinus or floor of the middle fossa encroaching upon this triangle, and it is therefore less reliable than that of Dench.

Middle Fossa.—In operating upon the mastoid two other points are of great importance—the location of the middle

fossa and the lateral sinus. The middle fossa may lie as low as the superior wall of the osseous meatus, or it may be considerably higher, but one never knows until the mastoid is opened and its configuration seen. So the possibility of its occupying a low position should always be borne in mind.

Lateral Sinus.—The lateral sinus lies on the cerebellar surface of the mastoid in a groove traversing the bone from above downward. The depth of this groove and the location of the sinus present great variation. The sinus may be found in some instances as far forward as the posterior wall of the canal or lie much farther back, say an inch in some instances, and between these extremes any position may be occupied. It varies also as to its depth from the surface of the mastoid. In some instances there is but a thin shell of bone between the sinus and surface of the mastoid. If in such a condition it lies far forward also, it is in danger of being wounded in the mastoid operation. Fortunately this configuration is somewhat rare, but must be borne in mind. In other subjects the sinus lies more deeply, so that numerous cells exist between it and the mastoid cortex, and between these two extremes all grades of variation occur. There are no landmarks upon the surface of the bone by which its position can be determined, so the mastoid should always be opened carefully as though the sinus occupied an anterior position until, as the operator progresses, it is found not to do so. The skiagraphic plates of the mastoid process afford some information as to the location of the sinus, but too much reliance must not be placed upon their readings.

Mastoid Process in Infants.—At birth the antrum and a few cells contiguous to it are found, but they apparently lie a trifle higher in the bone and much nearer the surface. The overlying cortex is much softer, so that in many instances a suppurative process will perforate the bone and the pus finds an exit on the surface of the mastoid rather than, by rupturing the drum membrane, appear

in the canal. The drum head lies on the surface and the canal is membranous. There is no tip, the facial nerve appearing on the external mastoid surface at the junction of its inferior and anterior border. The fossa subarcuata (or floccular fossa) exists as a well-marked depression beneath the superior semicircular canal (Fig. 57). This is lined with dura mater and through it bloodvessels pass to the mastoid antrum. In adult life this is obliterated to a mere fissure containing a process of dura mater and a small vein. Through this avenue infection may travel

Fig. 57.—Posterior and internal view of the temporal bone of an infant, showing the floccular fossa or fossa subarcuata. The dark opening to the left is the internal auditory meatus.

from the mastoid to the meninges. The parts of the bone are not so firmly joined in children as in adults and through these but slightly solidified fissures there are vascular communications which allow infection of the intracranial structures to more readily take place. The development of the mastoid occurs quite rapidly, so that before the end of the first year the osseous meatus can be made out as a distinct depression, while in a child between three and five years one is often impressed with the close resemblance to the adult bone in all respects except size.

MASTOIDITIS.

Definition.—Mastoiditis is an inflammation of the mastoid process. As might naturally be expected in a bone presenting so many variations in structure, the form which the inflammation assumes varies considerably both in its location and clinical manifestations. The bone is covered with periosteum and there seems no reason why a localized periostitis might not develop. While one can conceive that such a process could be due to causes outside of the ear, this is practically never the case, and as a subperiosteal accumulation of pus is so often seen secondary to middle-ear and mastoid involvement, in fact part of the process, it is as well to dismiss from one's mind these remote possibilities. As much may be said of primary involvement of the mastoid process, that is, not secondary to middle-ear disease. There seems no reason why the mastoid may not be the seat of an osteitis the same as any of the other bones. Cases are reported in which this is supposed to have occurred, the infection, usually tubercular or syphilitic, reaching the bone through the blood stream or lymphatics. As mastoiditis in tuberculosis and syphilis commonly arises from middle-ear infection, if the process in the mastoid was associated with middle-ear disease, it would naturally be attributed to it. The mastoid may also be infected from a penetrating wound or some other form of traumatism. In this case the process might possibly stop short of involvement of the middle ear and antrum, but it is probable that these cavities would as a general rule take part in the process. Moreover, the treatment in these very rare processes would be along the same lines as in those which are constantly coming under observation. Therefore it is sufficiently accurate to define mastoiditis as an inflammation of the mastoid process due to infection and secondary to involvement of the middle ear.

Causation.—The infection, as has been shown, usually reaches the middle ear through the Eustachian tube, although it may do so through a perforation in the membrana tympani. Moreover, as the path of infection is through the antrum, the process which causes the mastoiditis must involve the vault. This renders it necessary that the primary process should be a purulent one, either an acute suppurative otitis media or an acute exacerbation of a chronic process. Therefore the etiology of mastoiditis becomes the same as that of O. M. P. A. If the vault is involved, the antrum must be involved also, as they are only parts of the same cavity. It is probable that some of the mastoid cells contiguous to the antrum are involved in almost every case of O. M. P. A. in which the vault is invaded. Whether or not the process extends farther than this depends upon the promptitude with which drainage is established and its sufficiency, the character of the bone, the nature of the infection, and the resistance of the patient. The pressure of the accumulating secretion forces the infection farther into the mastoid cells if they are present. If the bone is sclerotic, the cells cannot be invaded, thus giving some relief to the pressure; so it not infrequently occurs that the pus escapes through the Rivinian segment and appears on the surface of the mastoid as a subperiosteal abscess.

Pathology.—The first stage of the involvement of the cells is a congestion and exudation of serum which may extend throughout the mastoid or be limited to a part of it. In this stage there is no breaking down of the septa between the cells. Politzer has shown that there are communications between the cells through which the process readily extends. The lining of these pneumatic spaces is a modified mucoperiosteum, but it is capable of great inflammatory reaction. If a mastoid is opened, which is in this stage of inflammation, no pus will be found, but the cells will be red and filled with serum which is usually more or less tinted with blood. Following this stage with more or less rapidity, according to the activity

of the process, is the formation of pus. If such a mastoid is opened, the cells are filled with pus but the septa seem intact. In the third stage, the septa between the cells break down and granulations are formed which are not osteoblastic in nature. One occasionally opens a mastoid where granulations have formed in cells with intact walls. They may be found in a part of the mastoid only, usually that part farthest from the antrum, while the rest of the mastoid is in any of the three stages above described. They are no doubt osteoblastic in nature and represent an effort at repair and limitation of the process by the formation of new bone. When they occur in a mastoid in which the remaining pneumatic spaces are filled with serum, it doubtless indicates that the condition is either in process of, or capable of, resolution. After the septa between the cells are broken down and an abscess formed, the pus exists in sufficient amount to render drainage through the tympanum practically impossible no matter how large the defect in the membrana tympani. Besides the process is of such a nature that the breaking down of bone seems to be a part of it. So as this pus accumulates it comes to the confines of the mastoid process and ruptures through them.

Location of Rupture.—According to location these ruptures may take place as follows:

1. Through the inner table. This structure in some bones is very thin and offers little resistance to the invading process. If the pus breaks through in the middle fossa, it produces what is termed an extradural abscess (also called epidural abscess); if into the posterior cranial fossa the condition is the same, only, as such abscesses are usually in the region of the sinus and have this structure as one of their walls, they are spoken of as perisinus abscesses. When the pus accumulates between the dura (or sinus wall) and the inner table, the external layers of the dura react by the formation of granulations. The size of these seems to depend largely upon the duration of the process, being found in some operations very small,

as though just beginning to form; in others, which have been long deferred, they are very large. In two patients upon whom the author operated and in whom the mastoiditis had existed for more than six months, these granulations were found to be over one-half inch in thickness, springing from the wall of the sinus and practically filling the mastoid cavity. The infection in one instance was the Streptococcus capsulatus, in the other the pneumococcus, but complete recovery ensued.

2. *Cortical Perforations.*—The pus in the mastoid may rupture through the cortex of the bone on the outer surface. This occurs more often in children. In adults the cortex is usually thicker and resists the process. Nevertheless, a cortical perforation in an adult is far from a rare occurrence. The most common location is directly over the antrum, but no part of the bone is exempt. The pus after finding its way through the softened bone accumulates beneath the periosteum, which it strips to a greater or less extent from its osseous attachment. One form of this perforation is through the cortex covering the zygomatic cells. In this form two types are seen. If the pus breaks through comparatively low and more posteriorly, it dissects the periosteum from the bone above and behind the ear, obliterating the fold between the auricle and the side of the head, thus forcing the former to stand away from the head. In the other type, the perforation being more forward and higher, the pus is prevented from spreading backward by the attachment of the temporal fascia and accumulates beneath the muscle in the form of a "deep temporal abscess."

3. Rupture may occur through the anterior surface of the bone, which is also the posterior wall of the external auditory meatus (or premastoid lamina) appearing in the canal as a subperiosteal swelling. In this, as well as in ruptures on the external surface of the bone, the skin and other structures overlying the periosteum may become inflamed and break down and the abscess thus discharge externally.

4. The rupture may occur on the inferior surface of the bone internal to the tip. In this event the pus accumulates beneath the muscles attached to the tip in the form of a deep-seated abscess (Bezold's type).

Symptoms.—Mastoiditis, being constantly associated with otitis media, it is often difficult to determine what part of the clinical manifestations is due to the mastoid involvement. It is well to consider every patient who has an O. M. P. A. affecting the vault as having mastoiditis also. The mastoid involvement may never pass beyond the first stage, its progress being checked by the establishment of drainage of the vault and antrum. It is then capable of resolution, while if it progresses to the formation of pus and the breaking down of cells, an abscess is formed and the process cannot resolve. Some authors call the first form mastoiditis and the second mastoid abscess, and it must be conceded that there is much in favor of this division.

Pain.—Pain may be considered as an almost constant symptom of mastoiditis. It varies greatly in the degree of its severity and in its duration. It may be nearly or quite absent in processes due to infection with the Streptococcus capsulatus, and is generally mild or absent in tubercular involvement of the mastoid. In the early stage of an O. M. P. A. before drainage has been established the mastoid involvement intensifies the pain. It is frequently excruciating and boring in character, and keeps the patient awake at night; but as spontaneous rupture takes place or the drum membrane is incised it usually begins to subside. Exceptionally, the establishment of drainage seems to have very little effect upon it. If pain is not relieved under these circumstances, it is fair to assume that the disease is progressing. Sometimes, after having subsided and remained absent or very mild for several days or a week, or even longer, pain again becomes severe. This is a symptom strongly indicative that the process has passed into the operative stage. In some patients the pain is milder, but they complain

of a vague discomfort in the mastoid region. This is more apt to be a later symptom, occurring usually in the operative stage.

Tenderness on Pressure.—Tenderness on pressure over the mastoid is one of the commonest symptoms of mastoiditis and may occur at any stage of the disease. It may be absent, as it frequently is in processes caused by the Streptococcus capsulatus. Before drainage is established the mastoid may be very tender, but this often diminishes together with the spontaneous pain after incision or rupture of the drum membrane. If the process is resolving, it gradually diminishes, being less from day to day until in a week or so it is absent; or, it may only remain a day or so. Or it may not appear until the middle-ear suppuration has existed for a week or more. It then has a far different significance than when it is an early symptom and is almost certain to be associated with mastoid abscess. A great deal has been written about the significance of the different locations of the mastoid tenderness. The author is not convinced that much of value may be inferred from the fact that the tenderness is in one place rather than in another. It is doubtless due to congestion of the periosteum and underlying bone and indicates that the process within the mastoid approaches the surface in the locality in which it is found. In a patient with a very thick cortex, it is more apt to be absent or come on later. Care should be taken in eliciting mastoid tenderness not to move the auricle or external auditory canal with the thumb which is applied to the mastoid, as this produces pain in inflammatory conditions of the meatus. In normal persons there seems considerable variation in the amount of tenderness on pressure upon the mastoid. Many subjects wince when firm pressure is exerted upon the tip; so not too much force should be used and the opposite side examined for comparison.

Fever.—In many adults there is very little rise of temperature. In others, there may be several degrees of

fever. It rarely rises above 102° F., and even at this point one would begin to suspect the presence of complications; while if it reached 103° F. one would look for its cause outside of the mastoid. It is different in the case of children. In them fever is the rule until the pus breaks through the mastoid cortex, when it may subside. A temperature of 103° or 104° F. in young children is not uncommon, while in infants it may reach 105° F. Oftentimes temperatures which are not due to the mastoid condition are attributed to it. A complicating pneumonia not rarely occurs in children and if it gives no signs a very puzzling situation may arise, especially if the question of operation rests upon the temperature. Every aurist of experience has been called to operate upon the mastoid of these little patients, on the mistaken idea that the mastoiditis was the cause of the fever.

Discharge.—The discharge sometimes gives one an intimation of the presence of mastoiditis. If it increases in amount so that it could not be produced in such a limited space as the tympanum and antrum, it probably comes from the mastoid. If it suddenly ceases, with an increase or no amelioration of the other symptoms, to appear again after a more or less extended interval, one suspects mastoid abscess. In some patients the character of the pus and the fact that it is flowing quite freely so long after the inception of the disease, cause one to suspect that a mastoid abscess has formed. This is especially true in capsulatus infections. The patient may not have pain or other symptoms, the pus being fairly abundant and of a peculiar creamy nature; upon bacteriological examination the prevailing organism is found to be the Streptococcus capsulatus. If this occurs in the third week or later, an operation is indicated; if in the second, an *x*-ray examination, if positive, indicates operation.

There is, however, a type of mastoiditis which may be considered as more or less subacute in nature, occurring usually in infants and children, in which the discharge continues very free into the fourth week or even later,

notwithstanding the removal of the adenoids and tonsils. Constitutional impairment arising from the suppuration may be present or apparently absent. When these mastoids are opened, they are usually found completely broken down, filled with pus and granulations, with the sinus plate and inner table intact. Tympanic drainage has apparently been adequate to prevent the occurrence of tension sufficient to cause rupture of the pus through the confines of the mastoid, although to increased tension may reasonably be attributed attacks characterized by a slight rise of temperature accompanied by restlessness from which these patients occasionally suffer. A mastoid operation is justifiable in this type of case, not only to relieve the patient from his symptoms, but also to prevent, if possible, the discharge from becoming chronic.

Closely allied to these are recurrent attacks of suppuration not infrequently observed in children. An attack of O. M. P. A. occurs and after a short period the discharge ceases. The membrana tympani may or may not become normal. Another attack occurs in from two weeks to three months and then another and so on, the nutrition and development of the child finally suffering to a considerable extent. While doubtless some of these processes are due to adenoids and tonsils giving rise to infection through the tube, many are caused by the persistence of a focus of infection in the mastoid, and, if relief is not obtained after removal of the adenoids and tonsils, a mastoid operation should be performed to remove this focus of infection

Fundus Changes.—There will be present in the membrana tympani characteristic changes of the accompanying otitis media. This will usually be all that may be seen during the first week of the disease. Later a certain characteristic fundus change occurs and may be considered as indicating with certainty that the process has passed the stage in which resolution is possible, in fact, indicates operation. This is a swelling of the postero-superior wall of the canal contiguous to the drum mem-

brane. The canal at this location overlies the antrum and the swelling is due to the process within the mastoid. In extreme instances, the canal may be so narrowed that a view of the anterior and lower part of the membrane becomes impossible. As usually developed the sagging is quite apparent when compared with the opposite side and extends into and includes the contiguous part of the drum membrane. Conical perforations are very frequently associated with mastoiditis in the later stages, and should always direct one's attention to the probability of mastoiditis. A condition to which Dr. Dench has called attention, having nearly the same significance as sagging of the posterosuperior wall, is a shortening of the canal from a prominent position of the drum membrane. This structure has a dull red, thickened appearance and while the natural depression of the membrane is absent, there is no local bulging. The whole membrane including Shrapnell's is moved toward the canal, giving the impression that the canal is not as deep as on the opposite side. It is produced by granulations in the tympanum and is a later sign usually indicating that the stage of resolution is passed.

Thickening Over the Mastoid.—Edema and inflammatory thickening over the mastoid is not infrequently seen and is due to the approach of the inflammation in the mastoid to, and involvement of, the cortex. In adults it usually precedes the rupture of pus through the cortex, but in children this often takes place without being preceded by noticeable edema or thickening. As other diseases may produce this condition, it becomes a valuable indication when they are excluded and then shows that the process has passed the stage at which resolution is possible.

Subperiosteal Abscess.—Subperiosteal accumulations of pus often occur, especially in children. In these swellings deep fluctuation can usually be detected. The skin and tissues over them may be red and inflamed or seem not markedly changed. When they occur above and behind

the ear, a characteristic appearance is produced. The auriculocranial fissure is obliterated and the ear stands away from the head (Figs. 58 and 59), and frequently seems lower than on the opposite side. If they are limited to the temporal fossa (deep temporal abscess), the ear may have this appearance but to a less marked degree, and fluctuation, while not so plain, can usually be elicited. If they occur more posteriorly on the mastoid surface or near the tip, the position of the auricle is not

FIG. 58.—Subperiosteal abscess. Anterior view. (Posey and Wright.)

changed so much, if at all. In Bezold's type of mastoiditis, the abscess does not remain confined between the bone and periosteum, but ruptures beneath the muscles attached to the tip, producing an oval ill-defined swelling in the neck adjacent to the mastoid.

Stiffness of the neck due to rigidity of the muscles attached to the tip is occasionally seen. Torticollis is a somewhat rarer symptom.

Diagnosis.—As mastoiditis is so constantly associated with an acute suppurative process involving the vault,

the diagnosis narrows itself down to determining in what stage is the process, and whether or not an operation is indicated. To recapitulate, the symptoms and signs given above are: (1) spontaneous pain; (2) tenderness on pressure; (3) fever; (4) change in the discharge; (5) fundus changes; (6) swelling of the soft parts and edema; (7) surface abscesses. It is not often that one is called upon to make a diagnosis upon any single one of these

FIG. 59.—Characteristic appearance in subperiosteal abscess adjacent to the external auditory canal. Posterior view. (Posey and Wright.)

symptoms and signs, although some of them are so characteristic that if present he may safely do so and test his diagnosis if necessary by operation. Spontaneous pain, tenderness on pressure, and fever, are the only ones in the list that are seen in the first stage when the process is capable of resolution, although they may be seen in the later stages and cause one to operate. These symptoms when they occur early, before drainage is established,

have a far different significance than when they occur in the second week or later, when they are often associated with fundus changes also. The duration of the process is dated from the appearance of pain or fever in the accompanying O. M. P. A. If a patient has a very tender mastoid at the time drainage is established, perhaps associated with fever and pain, and this tenderness gradually diminishes during the first week and at the same time the pain and fever become less, as they usually do, the process is resolving. No matter how tender the mastoid during the first week, if it is becoming less so, one need not operate. If this tenderness after diminishing for a week then begins to increase, or returns after being absent, then there is probably a process incapable of resolution. If one finds in addition a sagging of the posterosuperior wall, operation is necessary. If not the *x*-rays may throw some light on the subject. One does not feel inclined to operate on tenderness alone, although at times he may safely do so. It is a subjective symptom and is occasionally exaggerated by the patient. Moreover, in affections of the nerves supplying the mastoid integument hyperesthesia may be marked. One then is apt to find that the tenderness is not limited to the mastoid process. There is one type of case in which the pain is very intense and remains so notwithstanding a free myringotomy. When the mastoid is opened, the cells are found to be filled with blood. This hemorrhagic form of mastoiditis is not common, but an early operation is justifiable. Then again, fever may make an early operation the proper procedure. But one must satisfy himself that there is no other cause for the rise of temperature except mastoiditis, or some complication arising from it. At times the most skilful internist fails to find the cause of a rise of temperature, and to attribute it to the mastoid because the drum membrane does not appear normal or there is a discharge is manifestly unfair. On the other hand, one must not err in the opposite direction and delay operation if the mastoid is the cause of the fever. During

the first week in such an instance, when tenderness on pressure if present would be a great aid, it may be absent or its exact amount determined with difficulty on account of the patient being a child or infant. When the fever occurs later, other evidences of mastoiditis are apt to be present and the decision becomes easier. An infant or young child is very apt to develop a subperiosteal abscess early in the disease. This may indicate that the cause of the fever was in the mastoid. As compared with the number of operations upon subperiosteal abscesses in these little patients, those necessary when this condition is not present are probably in the minority.

If there is narrowing of the fundus from sagging of the canal wall, it will usually be associated with some of the other symptoms or signs, but if it occurs alone in an O. M. P. A. of ten days' duration, it indicates a mastoiditis incapable of resolution and therefore operation. A subperiosteal abscess is diagnosed by the elicitation of fluctuation; and if it occurs above and behind the ear, the characteristic position of the auricle, which stands away from the head, obliterating the auriculocranial fold. This appearance does, at times, occur with furuncle, but the swelling in furuncle is more apt to be edematous and pit on pressure and very rarely gives the sense of deep fluctuation. Movement of the auricle is painful in furuncle, and only rarely so in subperiosteal abscess. Moreover, examination of the meatus ought to enable one to decide. When the abscess is located upon the posterior canal wall, it may resemble furuncle very closely. (For differential diagnosis see Furuncle.) Edema or infiltration of the tissues over the mastoid, if one can be certain that the condition is due to a process within this structure, establishes the diagnosis of mastoiditis of operative type. Usually, however, one finds some of the other signs or symptoms present, such as narrow fundus, profuse discharge, etc. Every swelling of acute nature occurring in the mastoid region should be looked upon with suspicion and investigated by operation in case of

doubt. An enlarged broken-down gland often resembles the tumefaction due to mastoid disease. These inflamed glands do not usually cause the auricle to stand away from the head so markedly, and they are more apt to be situated lower than the condition resembling them due to mastoiditis. The proper procedure is to make an incision through them to the bone, then if the pus is beneath the periosteum, open the mastoid; if this membrane is normally attached, refrain from doing so unless for some other reason the operation is indicated.

The author has reported[1] four cases in which the middle ear was apparently normal and yet mastoiditis was present, the infection probably passing through the tympanum and reaching the mastoid, the membrane in the additus swelling and separating these cavities, after which the process continued in the mastoid while the middle ear cleared up, so that when coming under observation it appeared normal. In three of these patients a subperiosteal abscess indicated the nature of the process; while the fourth patient was seen in a dying condition and the autopsy showed the presence of sinus thrombosis and meningitis secondary to mastoiditis. In all patients where the diagnosis of mastoiditis is at all doubtful, an *x*-ray plate should be made and interpreted by one who has experience in this work. Mastoiditis occurring as an acute exacerbation of a chronic suppurative process always indicates immediate operation.

Prognosis.—In a great many patients who have mastoiditis in the early stages of an O. M. P. A., the process gets well during the first week or so. Some, however, pass into the stage in which resolution is impossible and operation is necessary. But the mortality of the operation in uncomplicated cases is very small. When sinus thrombosis, meningitis or brain abscess are caused by the mastoid infection the prognosis naturally becomes grave. The outlook is influenced by the general condition

[1] Annals of Otology, St. Louis, June, 1911.

of the patient as well as the nature of the infection. Diabetes with between 3 and 4 per cent. of sugar, especially if the urine contains acetone, makes the prognosis very serious. These patients seem more apt to have Streptococcus capsulatus infections. This germ is considered to indicate that the process is more grave, and this is to a certain extent true. Still the large majority of the patients with this infection make good recoveries. On the whole the prognosis of mastoiditis is good, but, in view of the ever-present danger of sinus thrombosis, meningitis and brain abscess, one should be more or less guarded in expressing an opinion.

Treatment.—The best preventive treatment is an early and free incision of the drum membrane, the cut extending into Shrapnell's. If this is done the inflammatory process, as far as possible, is minimized, perhaps only a few cells contiguous to the antrum becoming involved. A free incision of the membrana tympani is also the best treatment to be adopted after symptoms referable to the mastoid have developed. If spontaneous rupture has occurred and drainage does not seem sufficient, a myringotomy should be done. In these incisions after mastoid symptoms have declared themselves, the knife should be passed well into Shrapnell's membrane and withdrawn on the posterosuperior wall dividing the tissues to the bone for about one-fourth inch from the membrana tympani. This is practically the only treatment from which much may be expected. It is well, no doubt, to put the patient to bed and order a cathartic. The discharge is syringed from the canal as in O. M. P. A. It is well to determine in every patient the nature of the prevailing germ of the infection; also, to keep a chart of the temperature. The patient should be examined from day to day to note the progress of the disease. If thought desirable, cold in the form of the ice-bag or Leiter coil may be applied. It will frequently make the mastoid less tender and relieve the spontaneous pain but probably has very little influence for good upon the disease. All

are agreed that cold applications should not be persisted in for more than twenty-four hours, as by masking the pain and tenderness they serve to keep one in ignorance of the progress of the disease. The same may be said of any of the preparations of opium. Pain being one of the symptoms by means of which one determines whether or not the disease is improving, it is very unwise to mask it in any way, as its absence or mild character, due to the anodyne, might cause an unjustifiable postponement of operation. The use of cups, after scarification, or of leeches, artificial or living, is only to be condemned. They are probably of no value and only interfere with the proper elicitation of mastoid tenderness by the soreness which they cause. One cannot tell how much pain on pressure is due to the disease and how much to the treatment. For the same reason all applications which render the parts sore, such as rubefacients, blistering ointments and other remedies, should be absolutely prohibited. The Baer hyperemia treatment is probably without value. One must remember that an abscess is forming in bone and that nature has provided for a certain amount of drainage which must be encouraged and increased if possible. If this drainage is sufficient, combined of course with good resistance on the part of the patient, the process will resolve. The surgeon after seeing that tympanic drainage is as free as can be made, simply waits, as nothing which he can do further can reasonably be expected to influence the disease. He must bear in mind the proximity of vital structures and the danger of complications, and open this bony abscess as soon as he becomes satisfied that resolution is impossible or complications occur or seem imminent.

MASTOID OPERATION (SCHWARTZE).

Instruments.—There is a great deal of variation in the number and design of instruments required by different operators in performing the mastoid operation. While

it is always well to have an instrument at hand when needed, too many are to be avoided both on account of the expense and the fact that finding any instrument required on a table too well supplied with them only takes time and trouble. The following list comprises the ones the surgeon is almost certain to need before the operation is finished, and they are also sufficient to enable one to perform any mastoid operation properly.

1. A knife for the incision. A full-bellied scalpel of good size is the best type.

2. A number of hemostat forceps. Between six and twelve will be required.

3. A periosteal elevator. These are made hoe-shape, but the form shown in the cut (Fig. 60) will be found

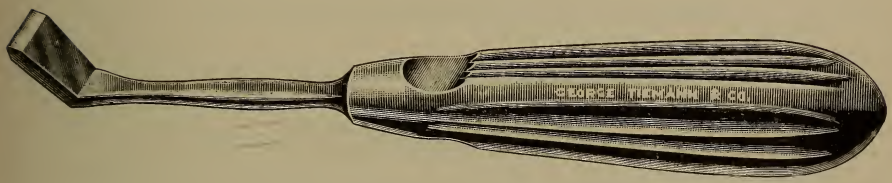

Fig. 60.—Author's periosteal elevator.

preferable. With it one can elevate the periosteum either forward or backward, and it is very useful in separating the muscles from the mastoid tip.

4. *Retractors.*—If the surgeon can have an assistant to hold retractors, those designed by Dr. Whiting are the best. If not, one of the self-retaining retractors such as Alport's or Yansen's are very useful (Fig. 61).

5. *Gouges.*—A set of three gouges designed by the author are advised. They are sharpened by a bevel on the anterior surface, which enables any desired thickness of bone to be removed without previously setting them as in instruments bevelled posteriorly (Fig. 62).

6. A mallet. These are made of rolled raw hide or lignum vitæ.

7. A set of three curettes (Spratt's). The larger, medium

and smaller-sized curettes (Fig. 63). The largest size is very safe to use when curetting the cells lying on the inner table either in the middle fossa or over the sinus

FIG. 61.—Yansen's self-retaining mastoid retractor.

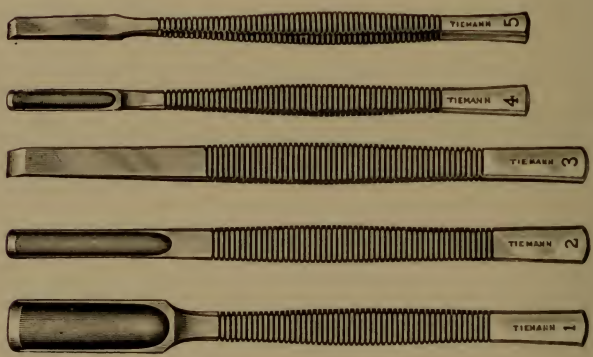

FIG. 62.—Author's mastoid gouges and chisels.

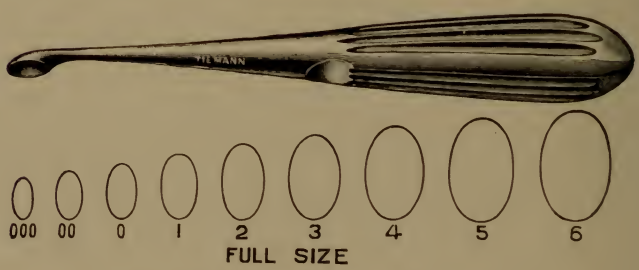

FIG. 63.—Spratt's mastoid curettes.

plate; the medium in breaking up individual cells and curetting in the depth of the mastoid between the sinus and posterior canal wall. The smaller one is useful in curetting around the antrum and zygomatic cells in the manner to be described.

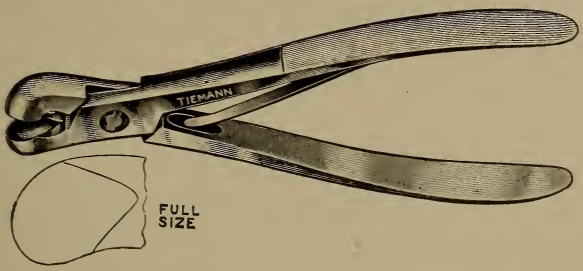

FIG. 64.—Mathieu's rongeur forceps.

8. Three pairs of thumb forceps. One plain-pointed, for the assistant to use in sponging and in picking the chips out of the wound, as they are made by the operator's gouge or curette. The other two, mouse-toothed, for approximating the edges of the wound.

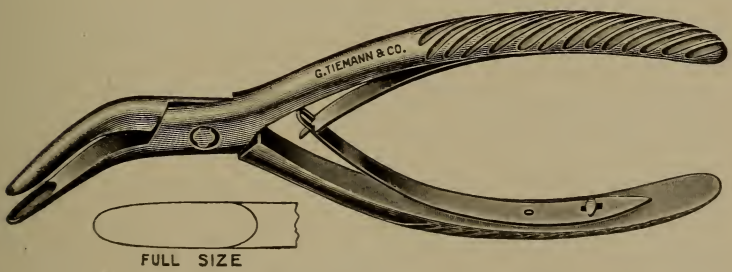

FIG. 65.—MacKernon's rongeur forceps.

9. Two pairs of rongeur forceps. One Mathieu's (Fig. 64) to remove overhang as it is formed by removal of underlying cells with the curette, and also to remove the tip. One MacKernon's (Fig. 65), which will be found

useful when it is necessary to remove the inner table while enlarging an exposure of the dura or sinus.

10. Two probes—large and small size—and a grooved director.

Fig. 66.—Scheibel's suture forceps.

11. A pair of scissors curved on the flat—used principally in freeing the tip.

12. Michel's clamps and forceps for placing them in position (Figs. 66 and 67).

Fig. 67.—Michel's metal clamp suture.

Preparation of Patient.—When possible, it is always better to prepare the patient for operation by a cathartic the night before and a light breakfast and no lunch, if the operation is to be performed in the afternoon, or without any breakfast, if in the forenoon. If local anesthesia is used, these precautions in diet are not necessary. If a diabetic has an acute purulent otitis media, it is well to put him on a diet at once, that he may better stand the mastoid operation should it become necessary. Unfortunately, but too often, the patient comes under observation when an operation is imperative and no time for preliminary treatment is afforded. Several hours before the operation the parts around the ear are shaved, thoroughly scrubbed with soap and water, washed with bichloride solution, then with alcohol. The ear is thoroughly syringed with bichloride solution, 1 to 5000, and a piece of sterile packing inserted. The parts are then covered with a sterile dressing. When

the operation is an emergency one, these preparations are made immediately before the patient is placed upon the table. At the New York Eye and Ear Infirmary patients are continually presenting themselves at the afternoon clinic in such a condition that an immediate mastoid operation is imperative and they are frequently admitted to the wards, prepared, and the operation begun within an hour from the time they have come under observation.

Anesthesia.—A general anesthetic is regularly used. While it is perfectly possible to perform a mastoid operation under local anesthesia, it has never become at all popular. The shock caused by the impact of the mallet on the gouges, the scraping with curettes and the crunching of the rongeur, all produce an effect upon the sensorium of the patient which more than offset any advantage derived from local anesthesia. Gas and ether are usually used, although if there are any pulmonary complications, gas and oxygen is safer. The patient may be anesthetized after being placed on the table or in an adjoining room and placed upon the table after going under.

Position of Patient.—The patient being on the table and under anesthesia, a sand-bag is placed beneath the neck and side of the head, the face being directed toward the side of the table corresponding to the good ear, the bandages are removed and a double towel slipped under the head. The two ends of the upper towel are then brought up over the head overlapping each other and pinned. A towel folded lengthwise is then placed in front of the ear, reaching from the one above to which it is fastened to well on the chest. Another posteriorly diverging from this one sufficiently to expose the field of operation. Immediately below the ear, and connecting these two, another towel is placed. The field of operation is painted with tincture of iodine and wiped with gauze saturated with alcohol. This treatment of the field seems to have an influence in preventing erysipelas and skin infections. The anesthetist sits at the side of

the table well out of the way. The surgeon stands at the head of the table and his first assistant, who is to sponge, at the side, during the first stages of the operation; later they may change places if necessary. If an assistant holds retractors, he stands between the operator and anesthetist. The instrument table is just back of the

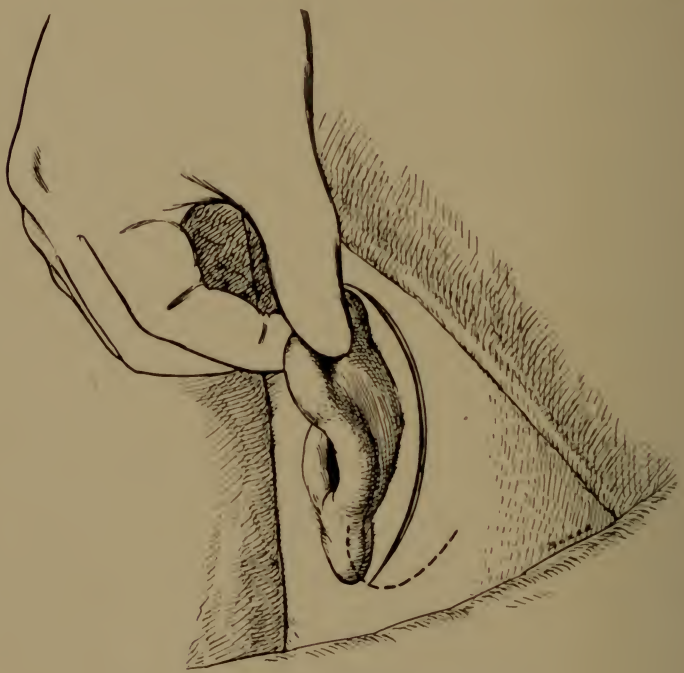

Fig. 68.—Incision in the mastoid operation.

space between the operator and his first assistant and convenient to both.

Incision.—The position of the lower end of the mastoid tip is located by palpation and the incision is made beginning at this point and extending to above the auricle, passing about one-fourth of an inch posterior to this structure (Fig. 68) and curving forward at its upper end.

The incision is made to the bone in its middle part. Here the bone is practically subcutaneous, while at the upper end it is covered with the temporal muscle and fascia, and at the lower end to a greater or less extent by the muscles having their insertion at the tip. If the incision is not curved forward at its upper end, it passes directly into the temporal muscle, and this structure will be needlessly cut; and besides, it may be very difficult to secure sufficient exposure to remove the zygomatic cells.

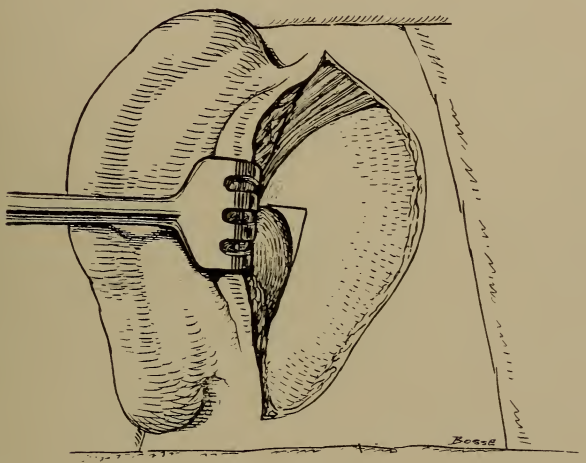

FIG. 69.—Anterior flap elevated and held forward with retractor. The Dench triangle is marked upon the bone.

Exposure of the Field.—The bleeding points are now caught with hemostats and with the periosteal elevator the anterior flap is separated from the bone and pushed forward until the posterior and part of the superior borders of the opening of the bony meatus are brought into view (Fig. 69). Care should be taken not to make a perforating wound of the membranous canal. The anterior retractor is now inserted. The temporal muscle is pushed up with the elevator, thus affording a view of the lower part of the squama.

Exposure of the Tip.—The tip is now "freed" by detaching the muscles inserted to it. This may be done with the elevator by scraping the muscular attachments from the bone, or scissors curved on the flat may be used. In either method, one works downward and inward carefully so that after the muscles are separated the instrument does not go sufficiently deep in the neck to touch the facial nerve. There is more danger of this accident while separating the structures attached to the anterior part of the tip. The facial nerve is very rarely wounded in this location but the possibility of this occurring is to be borne in mind. That the tip is thoroughly "freed" may be ascertained by the fact that its apex can be felt by the finger passed beneath it.

Posterior Incision.—The posterior incision may be made at this stage as advised by Whiting, or may be deferred until later when it is discovered that more room is needed. It is to be made on a level with the auditory meatus and extends from the margin of the posterior flap toward the occiput for from one-half to one inch, according to the room needed. The only objection to making it early is that in elevating these flaps the mastoid emissary vein is sometimes torn and leads to troublesome bleeding. The posterior flaps are now elevated, but the retractor is not inserted until during the operation it is found necessary.

Inspection of Cortex.—The surface of the bone is now inspected. If there is a subperiosteal abscess, the avenue of exit of the pus from the mastoid is to be found. This may be a cortical perforation on almost any part of the mastoid surface, in the zygomatic region or on the posterior canal wall through the premastoid lamina, or the pus may have dissected its way out through the Rivinian segment. When no subperiosteal abscess is present, the mastoid surface often presents a shaven beard appearance, being covered with little bleeding points usually indicating that an active process is going on within.

Removal of Cortex.—The next step is to remove the cortex and thus open the mastoid. In doing this a large-sized gouge is used and shavings of bone removed until the cells are encountered or pus wells up. If there is a perforation in the mastoid cortex, the removal of bone is begun from it, working in a direction downward and forward. It can readily be seen that the perforation may lie on the confines of either the sinus or dura, and if bone is removed upward or backward these structures

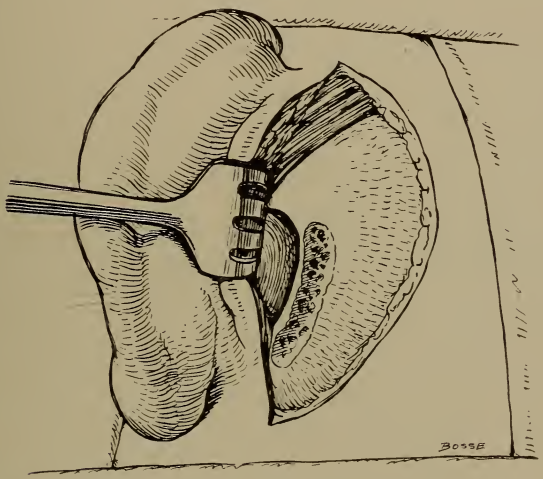

FIG. 70.—Mastoid cells opened upon completion of the groove along the posterior border of the canal.

may be endangered. This is to be avoided by adopting the plan advised above. If no cortical perforation is present, the best plan is to begin the removal of bone from the Dench triangle downward, keeping near the posterior wall and ending the cut in the cortex of the tip (Fig. 70). If the mastoid is a pneumatic one, and processes in this type of bone are those usually requiring operation, the second or third shaving in this gutter will open the cells or permit the exit of pus, although at

times the first shaving will do this, depending on the thickness of the cortex. Once the cells or the cavity of the mastoid is opened, one determines the topography of the individual mastoid upon which he is operating. He then proceeds to remove the remaining cortex insofar as possible without endangering the sinus or dura (Fig. 71). In removing the cortex, as well as using the gouge in any other part of the mastoid operation, it should be directed parallel with the sinus or dura and should be well under control. That is, it should not point in the

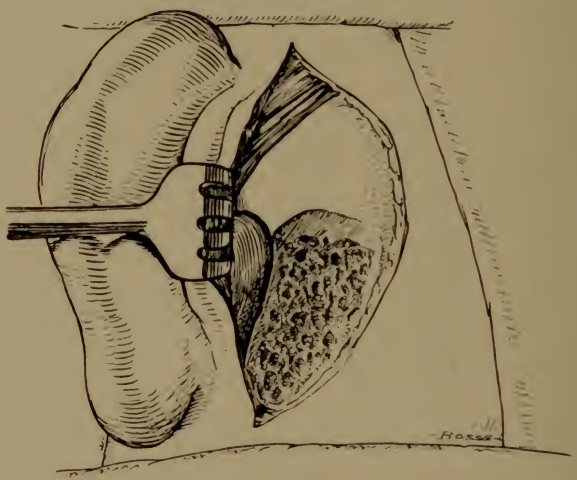

FIG. 71.—Cortex completely removed.

direction of the sinus or dura so that should it go farther than intended upon being struck with the mallet, these structures would be in danger of being wounded. If, as the cortex is being removed, pus wells up in greater quantity than could be contained in the mastoid cavity, one may safely assume that there is either a perisinus or extradural abscess with these structures exposed, and therefore exercise greater care not to wound them. It is always well after removal of the cortex, if an abscess cavity has been opened, to sponge it thoroughly and care-

fully, and inspect its walls. One by this procedure frequently derives an early knowledge that the sinus is exposed, and thus is in a position to avoid wounding it.

Removal of Tip.—It is often a good plan to remove the tip at this stage. For this purpose the gouge may be used, but the author prefers Mathieu's rongeur. One blade of this instrument is forced beneath the tip, taking care to hug the bone closely, and the piece grasped removed. If any soft parts are taken they are to be cut off with the scissors at the rongeur jaws. If cut farther from the jaws, there is greater possibility of wounding the facial nerve. It usually takes several bites of the rongeur to thoroughly remove the tip. That this is done is shown by the exposure of the digastric muscle, the fibers of which run forward and slightly downward.

Removal of Cells.—Frequently at this stage the inner table over the sinus or sinus plate, as it is called, can be made out. This is recognized by its color and contour. Its color is either white if the bone is thick, or of a slightly bluish color if thin and the sinus shows through. If it cannot be made out, the cells are removed with a curette until it is encountered, working upward and gradually and carefully backward from the lower part of the bone wound until it does come into view. In doing this work one good way of holding the curette is shown in Fig. 72. Grasped in this manner the instrument is under control; the wrist, being at rest on the parts around the wound, does not allow the point of the curette to slip and do damage. The thumb near the end of the instrument enables one to use great force safely. Another good method is to place the spoon of the instrument beneath any projecting edge of a cell wall and remove the parts in an outward direction by using the upper margin of the bone wound as a fulcrum. The side of the bowl being toward the sinus and the force exerted outward, this structure is safe. Some force may be used in an anterior direction toward the posterior canal wall. If, as the cells are being removed, working upward, the sinus

is accidentally exposed, or if there is a perisinus abscess, it is best to do the work around the antrum and in the other parts of the bone and come back to the region of the exposure at the end of the operation. The reason for this is that if the sinus is wounded it renders the rest of the operation more difficult, so it is advisable to postpone any danger of this accident until the end of the

FIG. 72.—One method of using the curette while removing mastoid cells.

operation. If no sinus exposure occurs, the cells are removed from the sinus plate behind to the posterior wall of the canal in front. If in doing this, there is an overhanging cortex, it may be taken away with the rongeur or large-sized gouge. Considerable variation in the mode of removing cells is not only allowable, but also advisable. The operator should study each manipulation which his ingenuity suggests in order to determine

that neither the sinus nor dura are endangered. Especially if a small curette is used, no force should be exerted in the direction of these structures, as the bone between the instrument and them may be thinner or less firm than it was believed to be and the instrument may easily penetrate them, perhaps carrying in its bowl much infectious material. A large-sized curette may be used to remove rough projections from the inner table over the sinus and dura, but even with this instrument but little bone should be taken at a time and the force used must be gentle.

The cells in the angle formed by the knee of the sinus and the inner table of the middle fossa are to be removed. They are sometimes very extensive in this location, and as they are taken away the inner table covering the dura above and the sinus below is mapped out. One must work carefully, as both of these structures are endangered.

The cells forward are removed, working along the inner table of the middle fossa until the antrum is opened. The cells existing in the posterior root of the zygoma and above the external auditory meatus are to be removed. The best method of doing this is as follows: the small curette is inserted into the antrum with bowl directed outward; then by using the posterior margin of the bone wound as a fulcrum, the spoon is made to travel outward taking the cells, and at the same time the instrument is rotated so that the back of the bowl becomes more and more directed toward the inner table, thereby assuring the safety of the dura. This procedure forms a cortical overhang which may be taken away with the rongeur or small gouge. The curette should never be introduced into the additus and removed scraping in an inward and downward direction, as it may catch the short process of the incus which lies in this situation and thus remove the ossicle.

Facial Nerve.—Sometimes the cells extend very deeply between the sinus plate and the posterior wall of the canal, and one feels that he is working in dangerous prox-

imity to the facial nerve while removing them. If these cells exist well forward and very deep, one should not curette their anterior walls; all posterior to them may with safety be removed until the internal table of the posterior fossa is encountered. In excavating these cells, the facial nerve is located by calculating its course from the horizontal semicircular canal. If this cannot be seen, as is often the case, it is to be remembered that it lies in the floor of the additus, the location of which may be readily determined with a bent probe. The nerve should lie internal to a line drawn downward from this canal parallel with the median line of the body. The prominence of the posterior semicircular canal is also about on this line and situated more posteriorly than the nerve.

Extradural Abscesses.—If an extradural abscess is found, the method of dealing with it depends upon the character of its dural wall. If this structure is covered with fine granulations, they are not to be molested. If, however, these granulations are very thick, as they are at times found to be in neglected cases, they may be removed. They sometimes spring from the sinus, nearly filling the mastoid cavity and may be mistaken for this structure until carefully examined. One may remove them if care is taken not to wound the sinus. In either event, the inner table is to be removed until healthy dura is encountered. This may be recognized by its color and general appearance. In removing this inner table the MacKernon rongeur forceps will be found useful. One jaw is inserted between it and the dura or sinus wall, hugging the bone, and before closing slightly withdrawn. This prevents nipping the dura which may be folded between the jaw and the bone. Some operators prefer the curette. If it is used, the same care should be taken to avoid nipping the dura. When the operation is completed, the cavity usually resembles that shown in Fig. 73, with the exception that the sinus should not be exposed.

Injury to the Sinus.—If at any time during the operation the sinus is wounded, the hemorrhage may be temporarily arrested by the operator placing his finger over the wound, then as he removes the finger iodoformized gauze is packed firmly against the sinus in the region of the wound. It is well, while working in the neighborhood of an exposed

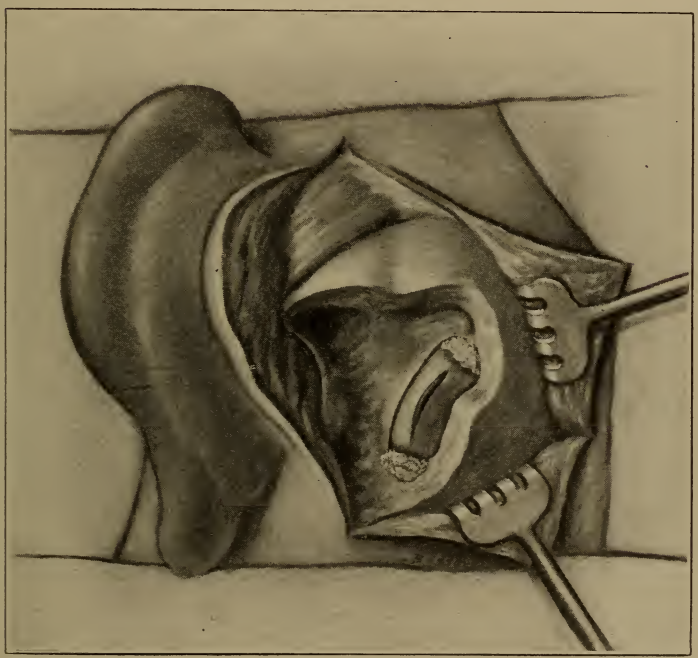

FIG. 73.—Operation for sinus thrombosis. The gauze rolls are in position and the sinus incised. The figure also shows the completed mastoid operation.

sinus, to have the gauze ready and the assistant on the alert, so that should bleeding occur the gauze may be rapidly placed in position. In some instances the gauze may be held by pressure and the operation completed, or if the operation is practically completed, the wound may be packed and the dressing applied. This method

of treating a wounded sinus may not lead to its obliteration. It may be possible after the hemorrhage is controlled to insert the gauze rolls as described under Sinus Thrombosis. If these are inserted, it leads to the obliteration of the sinus. They are usually, however, more convenient in the subsequent dressing of the wound. The subsequent course of a patient with a wounded sinus depends largely upon whether or not infection is introduced into this vessel. The author has wounded the sinus on three occasions without noticeable symptoms following, but he personally knows of other instances in which the accident has been followed by grave consequences, even death in some cases.

Injury to the Dura.—If the dura is wounded, unless the opening is already sufficiently large, which is seldom the case, the wound is to be enlarged by a linear incision of the dura passing through the puncture and iodoformized gauze packed in contact with the brain. This provides for drainage and aids in preventing meningitis.

Dressing.—The operation being completed, the posterior incision, if one was made, is first sutured; then the upper end of the original curvilinear incision is closed sufficiently to cover the surface of the squama which has been laid bare. Further closure is at the option of the operator, but the opening is usually left about one inch in length. Michel's clamps are excellent for bringing the flaps together. The wound is packed with iodoformized gauze, a sterile gauze strip placed in the canal, a circle of fluffed gauze is placed over the ear so that the auricle will not be cramped. Then a fluffed gauze pad is applied over all and upon this, absorbent cotton, and the whole retained by a bandage. It is important that the bandage should cover the cotton and draw firmly against the cheek and neck, especially in children who are apt, if the bandage is loose in this location, to insert their fingers and cause infection of the wound.

Mastoid Operation in Infants.—The mastoid operation in infants varies somewhat on account of anatomical

conditions. As the facial nerve is superficial, the incision is started more posteriorly below. As there is no tip, this structure cannot be removed. An attempt to remove what seems to be the tip only results in endangering the facial nerve. There is no osseous canal at birth, the auricle lying upon the squama. For this reason after elevating the flaps one often finds the exposure much higher than he supposed it would be, while making the incision. As there is no osseous meatus, one uses as a guide to the antrum either the posterior horn of the tympanic ring or the attachment of the canal to the ring, the antrum being located by lines similar to those in the Dench triangle. The antrum is much more superficial than in adults. An accident which may happen is that, when elevating the flaps the canal may be torn from the tympanic ring. If this is done, one might mistake the red thickened drum membrane for a cortical perforation. Merely to bear this possibility in mind is sufficient to avoid the mistake.

As the soft parts are often thickened in these little patients, it is frequently quite difficult to obtain a good exposure of the field and make out the landmarks. This should be done deliberately and with no haste, as the bone work is soon done once it is properly begun. In infants at times the squama is very soft, and it is possible to cut it with the knife when making the original incision, so one should not expect the knife to meet with the same resistance as in adult bones. Care should also be taken while separating the periosteum from these soft bones, as the elevator may be forced through the bone and this structure be separated from the dura or the latter wounded. If the child is older, there is a slight shelving near the tympanic ring, marking the beginning of the development of the osseous meatus and the antrum may be located as in adult cases. Very little work with the gouge is necessary in young children. A small shaving of bone is removed over the antrum, but the groove should not be made as in adults. After a small piece of bone is taken from the cortex, the rest of the operation may be

finished chiefly with the curette. The zygomatic cells are to be removed, as they are frequently developed out of proportion to the other cells.

Secondary Operation.—It occasionally becomes necessary to perform a mastoid operation upon a patient who has previously had this operation done. While in the main the procedure is the same as in the primary cases, one point demands attention. It may be that the surgeon knows how much of the sinus or dura was exposed at the previous operation. If he is not so fortunate, great care must be exercised in making the incision and exposing the field. The knife cannot be pressed down until bone is encountered, as it may open the sinus or dura. Usually one may cut to the bone above the auricle and also near the tip. Working from these points, with careful dissection with the scalpel aided perhaps with the curette, the surgeon exposes the field and locates the landmarks. Then the granulations, necrotic bone, etc., are removed upon the same principles as in the primary mastoid operation.

After-treatment.—The outside dressing is usually changed on the day following the operation. The clamps are removed in two days and the packing is changed in four days. After this, the wound may be dressed daily for a week or so, after which it may be allowed to go two days if the secretion is scanty. At each dressing after the first week or ten days, the wound is wiped with the cotton applicator, saturated with one-half strength peroxide of hydrogen (10 volume solution).

Temperature.—The temperature usually has a post-operative rise on the following day, not as a rule higher than around 102° F. If the temperature stays up or rises two or three days after the operation, the packing should be changed to ascertain, if possible, the cause. This may be infection of the wound; if not, further search should be made. (See Sinus Thrombosis and Meningitis.) A thorough physical examination is made of the patient, as the cause of fever may not be directly connected with the mastoid infection.

Granulations.—If the granulations become too exuberant, tincture of iodin may be applied after the parts are cleansed with the peroxide solution. As a rule these granulating wounds should be lightly packed, taking care that drainage is good. If the granulations are interfering with drainage or are large and flabby along the margin of the wound, they should be removed with the curette or scissors and their bases cauterized with a fused bead of nitrate of silver. The secret of success in mastoid dressings is cleanliness and the ability to determine when granulations are doing their work properly and need encouragement, and when they are interfering with drainage or causing infection and demand removal. As the discharge decreases and the cavity becomes smaller, a patch may be worn in place of the bandage, or the small dressing which is now required may be held in place by painting with collodion. If all goes well, the wound heals in about six weeks. Occasionally the time is a week or so shorter, but it is much more frequently longer.

Delayed Healing.—At times it fails to heal, a sinus persisting that will not close. These sinuses usually lead into the antrum and tympanum. A frequent cause is the continuance of suppuration in the middle ear. One is not inclined, while performing the mastoid operation, to disturb the structures in the vault of the tympanum. A necrotic ossicle, local caries of the tympanic walls, granulations or infected mucous membrane in the tympanic cavity, may keep the discharge active and prevent the posterior sinus from closing. At times the middle-ear process may be prevented from healing by adenoids and healing takes place after their removal. The sinus will at times heal if swabbed with iodin and not packed, at others a strand of gauze carried well into the antrum seems to hasten its closure. If it persists in spite of all treatment, a secondary mastoid or a radical operation will be necessary. It may be difficult to decide which to do in any individual patient. A decision is usually reached by the condition of the middle ear, the

chronicity of the process and often the findings at the secondary operation.

Prognosis.—The prognosis of the simple mastoid must be considered good. If the patient has complications before operation, the outlook naturally is less favorable; and these will at times occur after operation and produce death. The mortality is probably not over 2 per cent. The author has performed about 300 simple mastoid operations and so far as known but two patients have died. The death in each instance occurred after the patient passed from observation, and the cause is not known.

Blood-clot Treatment of the Wound.—A method of dealing with the mastoid cavity that has a few adherents is the blood-clot method. It consists in completely closing the incision with sutures or clamps and allowing the cavity to fill with blood. This clots, and at times becomes organized, resulting in greatly shortening the time required for healing and also in the production of a very slight scar. It more often breaks down and after the wound becomes clean healing takes place as in the ordinary method. It is without doubt slightly more dangerous and has not come into general use.

CHAPTER VIII.

SINUS THROMBOSIS (THROMBOPHLEBITIS).

The anatomy of the dural sinuses may be learned from the study of the text-books on Anatomy. Otologists are mainly concerned with the lateral sinus, jugular bulb and vein, more rarely with the superior and inferior petrosal, and the cavernous sinus. When "the sinus" is mentioned without qualification the lateral sinus is always meant.

Definition.—The term "sinus thrombosis" is applied to an infective process involving the dural sinuses which results in the formation of infective thrombi. This definition does not cover marantic thrombi nor those which sometimes form in the mastoid region without apparent infection, and either become organized, obliterating the sinus, or float into the circulation, causing sudden death from pulmonary embolism. Day[1] has recently called attention to this type of sinus thrombosis and reported six cases in which the clot was found organized or in process of becoming so. In some the lateral sinus consisted of a hard cord which upon incision proved to be firmly organized tissue, while in others the process had not gone so far. These patients did not present symptoms which could be attributed to sinus thrombosis, nor did they show a bacteremia, the diagnosis being made at the operation upon the mastoid.

Pathology and Causation.—The process is a phlebitis affecting the sinus wall and extending to the intima, thus producing a clot of varying size, or the thrombus may result from injury to the vessel or the infection may extend through the veins connecting the sinus with the original

[1] Transactions of the American Otological Society, vol. xiii, part 3.

process. It is well to bear in mind the fact that the inflammation of the internal layer of the sinus wall is an essential part of the process, although this in itself is not the cause of the symptoms. An injury to this coat of the vessel will result in a clot being formed, but unless the reparative process is complicated by the invasion of bacteria, the clot will become organized and the wound thus healed without the formation of an infective thrombus or the presence of symptoms. Therefore, in an injury to the sinus a great deal depends upon the nature of the field in determining whether the process which results will be one of repair or of an infective nature. Penetrating wounds in a septic field are especially diastrous, while such an injury in a clean field may heal without complications. A non-penetrating wound in a septic field may heal kindly or infection may occur. When such an injury heals without symptoms, it justifies the assumption that the resulting hemorrhage washed the infective material away before infection could take place. Nevertheless, a wound of the sinus is an accident of serious nature, and may be the starting-point of a sinus phlebitis of severe type. In a perisinous abscess one usually finds that granulations have formed protecting the sinus. At times, however, before this takes place the inflammation extends to the intima, thus producing a clot of greater or less size. The lateral sinus is usually the one involved, but the superior or inferior petrosal may be affected or the infection may extend to the jugular bulb through the tympanic floor or the condyloid veins. The process usually extends from the lateral sinus into the jugular vein, but it may also extend backward toward the torcular, even passing this point and involving the sinus on the opposite side. Rarely, by extension through the inferior petrosal, cavernous sinus thrombosis takes place.

If a mastoid operation has been done and the sinus exposed, its examination may show no indication of its involvement, although phlebitis with a mural clot may be present; but if the sinus is totally occluded by a thrombus,

it is apt to be more firm on pressure and not so resilient, and if it has existed some time there may be discoloration of the sinus wall and surrounding dura. At times the thrombus may have broken down and an abscess formed, which may have ruptured through the sinus wall. Such an abscess might not cause general infection if it was thoroughly walled off by organized clot, but by rupturing internally could readily produce meningitis or cerebellar abscess.

Symptoms.—When a clot which has formed in the sinus breaks down through growth of bacteria and pieces float into the general circulation, symptoms are produced. The same result follows if a very small clot clinging to the sinus wall floats away. There is a sharp rise of temperature, usually preceded by a chill or chilly sensations. The rise reaches to between 103° and 106° F., or even higher in exceptional instances. It falls in an hour or more to normal if there are no complications, and may be followed by sweating. This typical temperature curve is no doubt caused by the reaction of the patient to the introduction into the circulation of septic material as above stated. The attack of fever and so forth may be repeated in a few hours or on the following day, or even later, according to the activity of the process. Between the attacks in the early stages of the disease the patient feels quite well and does not seem sick. As the disease progresses and metastases develop, the temperature does not fall to normal between the rises and the patient seems sick all of the time. Exceptionally, the temperature does not fall to normal even in the beginning of the disease. This will usually be due to the fact that a certain amount of the fever is due to other processes than the sinus thrombosis. Then the temperature is more or less remittent in type.

The lymphatics along the jugular vein may be enlarged and tender from the process having extended into the vein. If the disease is neglected, they may break down and form abscesses. Changes in the eye-grounds frequently occur, either before or after operation. They

are evidently due to increased intracranial pressure and are more apt to be present if the vein involved is a large one and the interference with the return flow from the brain consequently greater. Greissenger's symptom is sometimes present. This consists of an edema and swelling, over the mastoid posteriorly and extending into the neck, which is caused by occlusion of the mastoid emissary vein. Various symptoms of uncommon occurrence have been attributed to jugular bulb involvement affecting the pneumogastric, spinal accessory, or glossopharyngeal nerves as they lie adjacent to the bulb in the foramen lacerum posterius. As the disease progresses without treatment, general sepsis usually occurs with the formation, in various parts of the body, of metastatic abscesses.

After the lapse of a varying length of time after the first rise of temperature in sinus thrombosis, if a culture of the blood taken from the general circulation is made, colonies of bacteria will usually grow within twenty-four to thirty-six hours. The bacteria which are found to be growing are usually of the same variety as those found in the aural pus and after jugular resection in the vein also. While bacteremia may doubtless be present in other conditions, the fact that it occurs in a patient suspected of having sinus thrombosis becomes of great value in the study of the disease.

Diagnosis.—In forming an early diagnosis, two points are of supreme importance, namely, the temperature curve and the bacteremia. In typical cases the temperature curve is very much like that of intermittent fever, although the individual attacks of fever do not, as a rule, follow with such regularity as do the paroxysms of malaria. In the latter there is the chill, the rapid rise of temperature with the rapid fall, followed by sweating. These may all occur in sinus thrombosis, although the chill and sweating may be less marked. Fortunately, the examination of the blood excludes malaria; for if the plasmodia of this disease existed in sufficient numbers to produce

such a paroxysm, examination of the blood would readily determine their presence. If the temperature curve is characteristic and malaria is excluded, the surgeon is justified in operating after the second rise, provided that a thorough examination of the patient fails to account for the temperature in any other way. If bacteremia is present before the second rise, the surgeon should operate. It may at times be extremely difficult to be certain that a single rise of temperature, no matter how characteristic it may apparently be, is due to sinus thrombosis. So one would be inclined to wait for the result of the blood culture or a second rise before operating, unless from the operative findings in the mastoidectomy (usually injury to sinus) he had reason to expect that involvement of the sinus was especially probable to take place. In atypical cases where the other causes for the temperature are exhausted, one might be justified in exploring the sinus and removing the jugular vein, but if the blood culture was positive it would be imperative for him to do so. Occasionally the patient either comes under observation when metastases have developed or, what is still rarer, develop them in spite of the surgeon's vigilance. These secondary infections indicate with great certainty that sinus thromobosis is present, provided, of course, that no other cause is apparent or discoverable.

Prognosis.—The prognosis of sinus thrombosis is always grave. Many patients recover after operation, while from time to time instances of a spontaneous cure are reported. If the thrombus is well walled off by organized clot, it may break down and the abscess thus formed rupture, through the external wall of the sinus into the mastoid cavity. It is possible for such a process to resolve without further operation than a mastoidectomy. Sinus thrombosis is also a not infrequent cause of meningitis and brain abscess, and this must be taken into account in estimating the probable outcome. Probably a conservative estimate of the mortality is that from 15 to 25 per cent. of the patients die.

Treatment.—A great deal may be done to prevent sinus thrombosis by a careful operative technic. Some inflammations of the sinus wall which result in the production of infective thrombi are caused by wounds which the surgeon knows that he has made; how many are caused by injuries which do not result in bleeding it is naturally difficult to say. It is, however, very probable that this occurs much more often than is supposed. A little spicule of bone piercing the sinus wall, or rough handling of the sinus, may result in the introduction of bacteria to a sufficient depth in its wall to produce an inflammation which extends to the intima. The number of patients who develop sinus thrombosis varies greatly in the experience of different surgeons and may to a certain extent at least, without injustice, be attributed to their lack of taking proper care of the sinus during operation. If the sinus is involved before operation, these remarks naturally do not apply; nor is it intended to convey the impression that a sinus thrombosis developing after the mastoid operation is necessarily due to improper technic, but that it may be, cannot be questioned. The proper treatment for the disease when developed is operation. The surgeon cannot take the responsibility of any expectant plan of treatment. He owes it to his patient to insist upon operation, and that at the earliest possible moment after the diagnosis has been established.

SINUS OPERATION.

Sinus Exposure.—The sinus is exposed from the knee to as near the bulb as possible. If it has not been previously exposed, the best instrument to begin the work is a large gouge. With this one takes successive shavings from the sinus plate until the sinus is bare, then with a curette or MacKernon's rongeur forceps the removal of the sinus plate is continued.

Incision of Sinus.—When sufficient exposure of the sinus is effected, small pledgets of rolled iodoformized gauze

are inserted between the inner table and sinus wall (Fig. 73) at either end of the exposed vessel. Unless there is a firm clot, they are inserted with sufficient firmness to bring the sinus walls into contact with each other, that is, collapse the vessel. The external sinus wall is now incised longitudinally, as shown in the figure. In making this incision, care must be taken not to injure the internal wall, as this opens the subdural space and may lead to infection of the brain or meninges. The outer wall may be picked up with a mouse-toothed forceps, nicked, and the incision completed upon a grooved director, or with probe-pointed scissors. In this way injury to the internal sinus wall will be less probable.

Removal of the Clot.—If there is a clot in the sinus, it is turned out and the walls inspected. The lower gauze roll is now removed and if hemorrhage is free immediately replaced. If bleeding does not occur freely from this lower end, a dull ring curette may be introduced with the object of dislodging the clot which must be done if possible, especially if it extends into the inferior petrosal sinus. Under no circumstances is a sharp instrument to be used, as injury to the intima may be produced, resulting in phlebitis and extension of the process. If this maneuver succeeds in producing free bleeding from below, the gauze roll is replaced and the upper end dealt with in the same manner. If bleeding does not take place from the upper end after the pledget has been removed and the dull ring curette used, it indicates that the clot extends backward toward the torcular. This clot must be removed, as if it is allowed to extend the sinus on the opposite side may become involved. To accomplish this bone is removed over the line of the sinus, which has been ascertained by the direction of the dull curette when attempting to remove the clot. The outer wall of the exposed sinus is incised and the bone removal continued until free bleeding is obtained, even if it is necessary to extend the operation to the median line to do so.

Jugular Resection.—Indications.—If upon making the initial opening of the sinus a firm clot is found, and after its removal free bleeding is obtained from below, the jugular operation may be postponed if the patient seems weak or there is any other good reason for doing so. This is not advised, but one may justify oneself for doing so. If, however, no clot is discovered, or only a small one, the jugular resection must be done at once. If sufficient reasons existed to justify exploration of the sinus, nothing will be accomplished under these circumstances unless the jugular is resected, as there is in all probability a mural clot with phlebitis, and the process extending as it may beyond the limits of the exposed sinus is in no way influenced by the sinus operation.

There is no good reason why the jugular should not be resected before the sinus is explored. The instruments and hands of the surgeon and assistants are sterile and the necessity of resterilization before performing the jugular operation is avoided.

Operation.—The neck is prepared as for any surgical operation. The shoulders of the patient are placed upon a sand-bag to allow the head to fall backward and produce a prominence of the sternomastoid muscle. The incision is made along the anterior border of this muscle, reaching from slightly above the clavicle to near the angle of the mandible, and extends through the skin, superficial fascia and platysma myoides, and usually reveals the fibers of the sternomastoid muscle. If it does not, this muscle should be sought by pushing back the posterior margin of the wound with the handle of the scalpel. Having located the anterior border of this muscle, it is held back with a blunt retractor. An anterior retractor is not advised, as the relation of the parts is often obscured by its use. Throughout the rest of the operation blunt dissection is used. By retracting the sternomastoid muscle, the sheath of the vessels is exposed. The vein lies external to the common carotid artery (Fig. 74) and between the two, but on a deeper level, the pneumogastric

nerve. The nerve is not shown in the cut as it does not come into view until a later stage in the operation. At the lower part of the incision passing over the sheath, and therefore superficial to the vessels, is the anterior

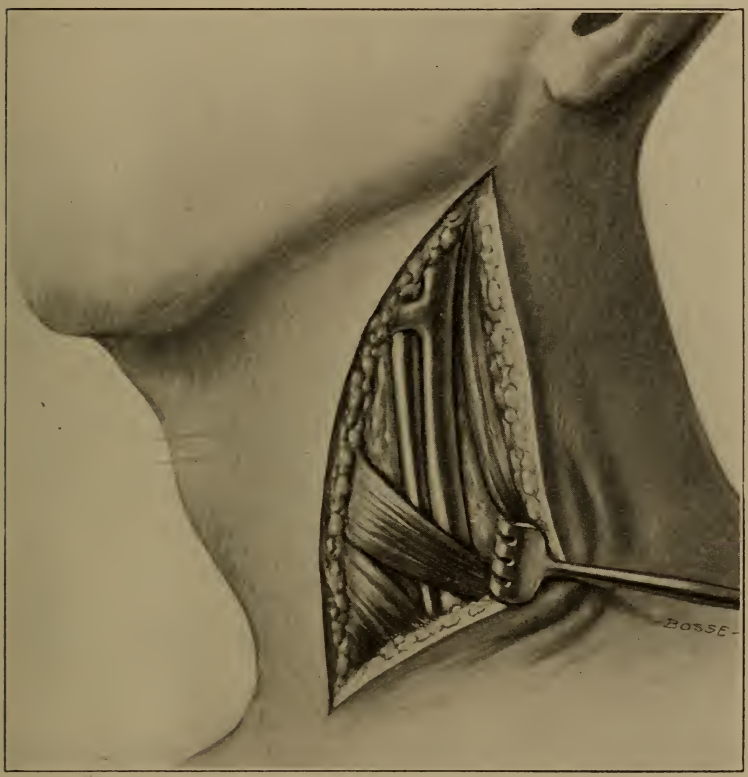

FIG. 74.—The vein and artery exposed in the operation for jugular excision. The pneumogastric nerve does not show, as it is covered by the artery and the vein.

belly of the homohyoid muscle. Lying in the sheath superficial to the vessels is the decendens hypoglossi. This may at times be seen but no attempt to preserve it is made. If from the appearance of the vein or any other reason a low resection is determined upon, the

homohyoid muscle may be divided on a grooved director. The sheath of the vessels is merely a thin layer of connective tissue and requires no division. If one conceives this sheath as tissue which must be passed through before coming in contact with the vein, he will often enter the vein in trying to do this. Disregarding the sheath, the vein is separated (with the handle of the scalpel) from the other structures with which it is associated. The artery is not disturbed as its location may readily be determined by its pulsation. Care is taken not to wound the vein. If this accident occurs, it should be caught in the forceps at the site of the bleeding. It is separated below and a double ligature thrown around it. Before tying these ligatures, the surgeon makes sure that the pneumogastric nerve is not included within them by drawing the vein outward and locating the nerve in relation with the artery in the bed from which the vein has been separated. Under no circumstances is the nerve to be handled or picked up with the forceps. The ligatures are now tied and the vein divided between them, and the upper end caught in a forceps to prevent hemorrhage in the subsequent steps of the operation should the ligature slip. The vein is now separated, working upward, and its anterior branches tied and incised between two ligatures. There may be three of these, corresponding to the thyroid, lingual and facial branches of the carotid, but the more common arrangement is for these branches to be represented by one large anterior trunk; at times, there are two. There are none posteriorly unless anomalous ones. The vein is separated to a little above this anterior branch or branches, and cut between two ligatures. In working in the upper angle of the wound, care should be taken not to injure the spinal accessory nerve. This may not be encountered if it lies behind the vein in its course to the sternomastoid muscle; but it may pass in front of the vein when it is to a slight degree endangered. This removes the vein and the pneumogastric nerve is more or less plainly visible (see Fig. 75). A strip of gauze may be

inserted in either angle of the wound for drainage, the two nearly meeting in the centre, and the skin incision closed with clamps and a sterile dressing applied.

After-treatment.—The clamps are removed in two or three days and the gauze drains shortened as fast as

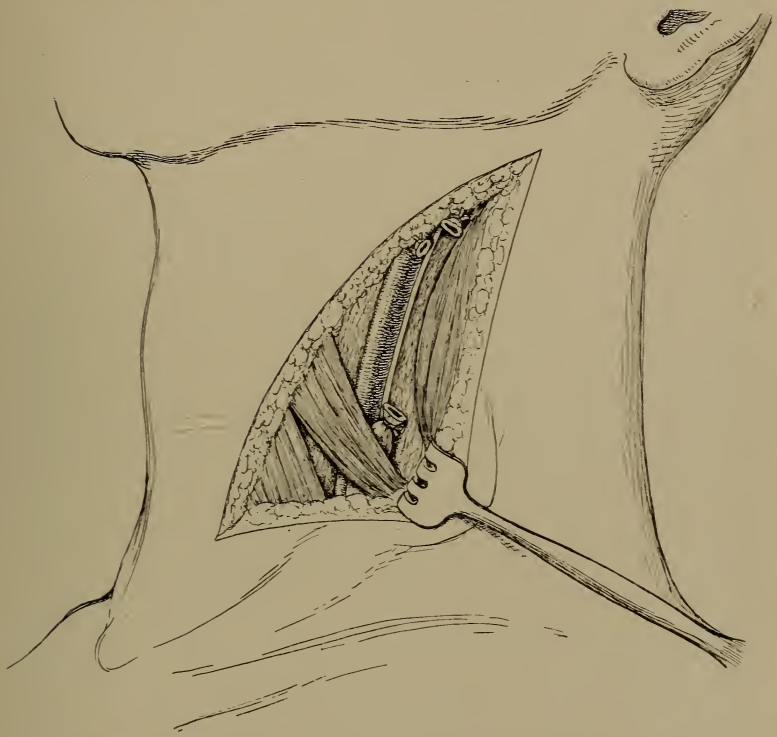

Fig. 75.—The jugular vein is removed thus allowing the pneumogastric nerve to come into view.

permitted by the necessity for drainage. The mastoid wound is dressed as in the ordinary mastoid operation with the exception of the gauze rolls which control the bleeding. The upper one very seldom causes any difficulty and may be removed in about four days without the occurrence of hemorrhage; however, should bleeding

occur, it must be reapplied and allowed to remain for a few days more. If there are no contraindications, the lower one may be left for five days. One must be prepared for bleeding when changing this and immediately insert a fresh one. Usually there is no hemorrhage at the second dressing two or three days later. Once the bleeding does not take place when the gauze rolls are removed, the wound is taken care of as after the mastoid operation. In the after-treatment of these patients, when after all that has been done, some sepsis remains, benefit may at times be obtained from the administration of an autogenous vaccine or the His leukocyte extract.

If the temperature does not fall after the operation, within a few days search should be made for a focus of infection which may have been left. This may be in the sinus toward the torcular, in the jugular bulb and inferior petrosal sinus, or rarely in the lower end of the vein which at the operation was not removed sufficiently low in the neck. Under these circumstances, the sinus must be explored posteriorly even to the median line if necessary or more of the lower end of the jugular resected.

Gutter Operation.—If the bulb appears to be involved, it may be opened by the gutter operation. This operation is performed by uniting the upper end of the jugular incision with the lower end of the mastoid incision. In doing this care should be taken not to wound the facial and deeper in the wound the hypoglossal which emerges just anterior to the vein. The bone is removed over the sinus, working downward until the external surface of the bulb continuous with the vein is reached. This removes the outer margin of the jugular foramen. The outer wall of the bulb may be incised and the clot cleared out. Great care should be taken not to wound the structures adjacent to the inner wall of the sinus or bulb in the jugular foramen, as in this location the ninth, tenth and eleventh cranial nerves are found. Usually removal of the clot from the bulb will result in free bleeding from the inferior petrosal sinus; if it does not no attempt should

be made to secure this result by introducing a curette. The gutter operation is a formidable procedure and requires for its performance an intimate knowledge of the anatomical conditions. Fortunately it is but rarely required.

BLEEDING SINUS.

Pathology.—Occasionally after a mastoid operation in which the sinus has been exposed, bleeding occurs during the healing process. There is great variation in the thickness of the outer wall of the sinus in different subjects. In some it is so thin that the blue color of the venous blood shows through very plainly; in others, so thick that the sinus wall does not differ in color from the dura in other situations. It is in these thin sinuses that hemorrhage at times occurs. The wall does not appear to be thick enough to throw out granulations and take part in the healing process, as does the ordinary sinus wall. The surgeon may have noticed at the time of operation that the sinus appeared thin. The gauze was packed around the sinus as in ordinary dressings. During the healing process this thin wall, softened by the effort to throw out granulations, becomes adherent to the gauze, and when this is removed at the first or some subsequent dressing, there is a gush of blood, which coming so unexpectedly is truly alarming.

Cases.—The author recently treated a patient who suffered from this condition. A girl, aged ten years, entered Dr. Dench's service at St. Luke's Hospital. Operation revealed a small perisinus abscess. The sinus wall was very thin. On the second dressing, five days after the operation as the house surgeon removed the packing, there was profuse bleeding which he immediately arrested by packing the wound with gauze. Every time the gauze over the sinus was removed during the ensuing three weeks profuse hemorrhage occurred. A small piece was therefore left at the location of the hemorrhage and changed about every six days, while the rest of the wound

was dressed daily. The patient had one rise of temperature (104.5° F.) on the day following the first bleeding, but as the blood culture was negative and no further rises occurred, the jugular was not resected. The recovery was complete. Two others have come under the author's observation, although not his personal patients. In one the history is practically the same as given above. In the other, the temperature remained of a somewhat remittent type, around 102° and 103° F., but as the blood culture remained negative, the jugular was not resected until a mestastasis developed, when this operation was done and a cure resulted.

These patients are in a different class from those in whom bleeding occurs at the time of the operation, for one is then expecting hemorrhage at subsequent dressings and is prepared for it.

Treatment.—The treatment is outlined in the description of the above cases. If the vein is not too friable nor the bleeding very profuse, it is best to insert the gauze rolls as in sinus thrombosis operations. These can be left for about six days if the temperature remains down and the rest of the wound dressed daily. If this cannot be done, one must be satisfied with a piece of gauze, as small as possible, over the sinus wound. This to be left for from four to six days, while the remainder of the packing is removed and the wound dressed as required daily or every second day.

CHAPTER IX.

OTITIS MEDIA PURULENTA CHRONICA (O. M. P. C.).

Chronic suppuration of the middle ear is a common disease and on account of the serious nature of its complications one of the most important also.

Etiology.—It usually follows and is a result of the acute form of suppuration. It is customary to consider the process chronic after it has existed three months. This is an arbitrary division but it answers very well. Some processes seem chronic from the outset, that is, develop without reactionary symptoms. Since chronic suppuration usually is a result of the acute form, the causes given for O. M. P. A. are operative in O. M. P. C. also. Lack of resistance, the serious nature of the lesions arising from the acute process, continuance of the nasopharyngeal lesion causing the acute disease (especially adenoids) are some of the influences producing chronicity. Middle-ear suppuration occurring in scarlatina, diphtheria, typhoid fever, and tuberculosis show a special tendency to become chronic. Chronic suppuration more often follows the acute form occurring in childhood, less often in adults.

Pathology.—The changes in the mucous membrane are as follows: Thickening, infiltration with round cells, development of new connective tissue forming adhesions or bands; the epithelium may lose its ciliated character. Granulations and polypi may develop. Necrosis of the ossicles or walls of the tympanum and antrum may occur.

Cholesteatoma.—Aural pus usually contains epithelial cells in varying numbers. When they become of the squamous variety and come from the tympanum and

antrum, and their free discharge is interfered with, or they are formed in excessive numbers, they accumulate in putty-like masses called cholesteatoma from the fact that these masses contain cholesterin crystals. The accumulations consist of epithelial débris or the cells may be arranged in the form of nests of greater or less consistency. The dermatization of the mucous membrane is usually brought about by the extension of the epithelium through a perforation; or it may in rare instances occur as a primary process, the ciliated epithelium becoming so changed that desquamation with accumulation of the scales takes place. As one often observes the inner wall of the tympanum dermatized without the process extending to the vault and antrum, it would seem that some change of the epithelium in the external auditory canal is necessary, or at least conduces to the formation of cholesteatoma. As the accumulation increases, and is unable to escape, pressure with erosion of the bone takes place. The entire mastoid cavity may become absorbed so that only the cortex remains, and even this may be perforated by the process. Or rupture through the inner table may occur and the masses accumulate between this structure and the dura which they press back upon the brain. The process may cause erosion of the posterior canal wall and the accumulation may escape through the meatus, and a cure may thus result. One not infrequently sees patients in whom this has taken place, the resulting cavity resembling to a certain degree that left after the radical operation. The cholesteatomatous mass may burrow beneath the skin of the anterior canal wall, eroding the bone and involving the temporomaxillary articulation. Nor is the ivory-like bone of the labyrinthine capsule free from the encroachment of these masses, as they not infrequently erode the horizontal semicircular canal or more rarely other parts of the labyrinth.

Symptoms.—There is regularly a purulent discharge in chronic suppuration of the middle ear, but there are

great variations in its amount and character. In some patients it is free, appearing continuously at the outer end of the meatus; in others, being so scanty that it dries, either forming a crust on the walls of the canal or a plug completely occluding its lumen. Some patients may be free from discharge for varying periods, generally short ones, the disease being in a latent state, but by no means cured. At times it is thin and watery, at others, creamy and frankly purulent. It may possess odor or be free from it. Odor is due to the growth of the germs of putrefaction and is usually present in pus coming from cholesteatomatous processes or when bone necrosis is present. The impression that processes which cause foul discharge are more serious is usually justifiable, although not always correct. The pus may be more or less bloody in character when polypi or granulations are present.

There is regularly more or less impairment of hearing which tests show to be due to disease of the sound-conducting mechanism. If the labyrinth is involved secondarily the characteristic tests of perceptive mechanism lesion will be present. At times patients come on account of their deafness, the discharge being so scanty that it has not annoyed them or at times even been noticed. The deafness is caused by lack of motility of the conducting mechanism, or to presence of secretion covering the labyrinthine fenestræ. In a few patients who had profound impairment of hearing associated with chronic suppuration Manasse found, postmortem, in their temporal bones the changes characteristic of otosclerosis.

Tinnitus is often an annoying symptom and at times severe. As a rule, however, these patients do not complain of it as much as those suffering from chronic non-suppurative processes. Patients with chronic suppuration of the middle ear are apt to be below par physically. They may have the disease on account of their poor resistance or their physical inferiority may be due to the long-continuing suppuration. There can be no doubt

but that this disease affords a focus of infection, the results arising from which are as yet scarcely known or appreciated. Children frequently show phenomenal improvement after being cured of the middle-ear process, their backward development being fairly attributable to some deleterious influence arising from the disease.

Facial paralysis occurs at times. It is due to necrosis of the bone containing the nerve, to neuritis or perineuritis caused by infection, usually through a dehiscence in the Fallopian canal wall, or to cholesteatomatous erosion and pressure.

Loss of taste on the anterior two-thirds of the tongue on the involved side is present in nearly 50 per cent. of the patients, due to involvement of the chorda tympani in the middle ear. In a small percentage there is also a loss of taste sense on the posterior third of the tongue, soft palate or fauces, which is attributed to involvement of the tympanic plexus as it lies on the internal tympanic wall.

Pain is not present unless there are complications or acute exacerbations; yet discomfort and intermittent pains are at times complained of. Vertigo should always lead one to suspect some labyrinthine involvement, although it sometimes occurs after syringing the ear, due no doubt to disturbance at the labyrinthine fenestræ or to the temperature of the solution acting as in the caloric test.

Signs.—In making an examination of the fundus of the external auditory canal, one endeavors not only to locate the loss of integrity of the drum membrane which usually exists, but to form some conception of the nature, extent, and location of the process, which will guide him in determining the prognosis and treatment. While wiping away the secretion its amount, character and source are noted, and in forming an opinion as to the lesion causing it, the length of time it has persisted and whether continuous or intermittent, has a distinct bearing. Perforations of the drum membrane in O. M. P. C. may be divided into three classes:

1. Large perforations of the membrana tensa permitting a view of the internal tympanic wall.
2. Small perforations of the membrana tensa.
3. Perforations involving Shrapnell's membrane.

Large Perforations.—Large perforations permitting a view of the internal tympanic wall present considerable variation: (*a*) the membrana tensa may be nearly completely destroyed, a small margin only of the membrane left along the tympanic ring, or this margin may be absent at some part of the segment; (*b*) only the central part may be destroyed when the perforation is usually more or less kidney-shaped, the manubrium projecting into what corresponds to the hilum of the kidney. This process may be either intact, in which event there is usually a substantial margin of membrane attached to it, or it may be more or less necrosed; (*c*) the posterior part of the membrane may be absent, the anterior part either not having been destroyed, or if this took place, it has reformed with cicatricial tissue. In total loss of the drum membrane (*a*) the manubrium may either be present or have sloughed away from disease. If it is absent, the process has usually invaded the tympanic vault. If present, it is usually adherent to the internal tympanic wall and at times seems much broader than normal. This is due to rotation of the ossicle so that the short process points more forward than normal. In large posterior perforations it usually lies in the anterior margin of the perforation attached to the inner wall of the tympanum. In these, as well as in those which involve nearly the whole of the membrana tensa, the long process of the incus attached to the stapes may be seen, or if the former has necrosed, the stapes alone may be present. One frequently sees these large perforations, which have become healed with cicatricial drum membrane, more or less adherent to the internal tympanic wall, or dermatized posteriorly and membrane anteriorly, the ear being dry. So they are not in themselves of serious import. Dermatization in a kidney-shaped perforation usually takes place by the mar-

gins of the perforation adhering to the internal tympanic wall which then dermatizes over the area of the perforation. When this occurs the ear becomes dry. These kidney-shaped perforations are more common with tubal involvement, and patients with them often give the history of intermittent discharge. The ear will be dry except for a few days or weeks in each year, or they may respond rapidly to treatment, as in the case of the patient whose fundus is shown in Figure 76. Nevertheless, the discharge may come from the vault and antrum in this type of perforation; still, unless contraindicated for some special reason, patients with this type of perforation are entitled to a thorough trial of non-operative

Fig. 76.—Kidney-shaped perforation. From a patient of the author. History of short attacks of suppuration between which the ear was dry.

treatment. The following remarks apply to classes *a* and *b*, that is, nearly total destruction of the membrana tensa or large posterior perforations. If the internal tympanic wall is dermatized and there is a purulent discharge, it must be coming from the vault and antrum, as the Eustachian tube is usually closed. A sinus will often be found leading beneath the posterior fold (Fig. 77). An odorous discharge in such a process seldom ceases without operation. Patients with this fundus condition are not infrequently seen who have resorted to all sorts of local treatment for years without relief. In operating upon them cholesteatomatous masses are not infrequently found. The internal wall may be dermatized and the

patient have an intermittent discharge, coincident with an acute nasopharyngeal process, but this is not common. In them the dermatized area over the tube breaks down with the tubal infection and discharge occurs for a short time only. The internal tympanic wall may be covered with mucous membrane which is reddened and thickened. It may be difficult at times to distinguish this from an inflamed tympanic membrane, and in fact incisions have been made under this misinterpretation. When the membrane is not markedly diseased, the pus may be coming from the Eustachian tube. One may be able to determine this by drying the cavity, then inflating, when,

FIG. 77.—Large perforation—marginal posteriorly. Continuous suppuration for years. Pus discharging through a sinus beneath the posterior fold. Internal tympanic wall dermatized. Radical operation showed granulations and cholesteatomatous masses in the vault and antrum.

if the tube is responsible for the discharge, it will be forced into view. Patients in whom this is the case are more apt to show recurrences, being well for periods varying from one month to a year, then having discharge for a few months or so. The attacks doubtless being caused by reinfection of the tube. The discharge is usually mucopurulent and without odor, and the process offers encouragement for the success of treatment directed to the tube and nasopharynx. If pus is not forced from the tube upon inflation, the otoscope may be tried, when, by rarefying the air, pus may be drawn from the vault and antrum. If this is the case, it will usually be found

that the discharge has been constant and possesses odor. Such a process offers very little encouragement for local treatment. Granulations may spring from the internal tympanic wall. If this is the case, an attempt may be made to relieve the process by local treatment, but it will usually be without success. The patient from whom Fig. 78 was taken was treated for six months without any relief. At the operation the foot-plate of the stapes was found necrosed, the ossicle lying in the mass of granulations. Polypi may spring from the internal tympanic wall or from the vault and appear in the canal. When they are present they must be removed before a view of the parts can be obtained. The occurrence of a polyp

FIG. 78.—Large posterior perforation. Granulations in the region of the oval window.

usually indicates that very little may be expected from non-operative treatment.

Small Perforations.—Small perforations of the membrana tensa allow no part, or at most a very small one, of the internal tympanic wall to be seen, so one must judge of the condition of the tympanum and antrum by the character of the discharge and the appearance and location of the perforation. These may exist with clean-cut edges or granulations may spring from their margins and more or less fill the opening, or forcing themselves through, produce a red prominence on the surface of the drum membrane. These granulations usually occur in processes which do not respond so readily to local treatment.

According to location, small perforations may be divided into several classes.

(*a*) Openings in the anterior part or near the center of the drum membrane. The discharge in these perforations is apt to be mucopurulent and more or less intermittent. The patient from whom Fig. 79 was drawn had discharge for about two or three weeks in each year for a number of years. Each attack occurred with an acute nasopharyngeal involvement and was, no doubt, due to tubal disease. One should treat a patient with the small central or anterior perforations nonoperatively until experience with the individual process teaches him that his efforts are fruitless.

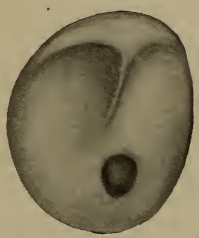

FIG. 79.—Small central perforation.

(*b*) The perforation has a different significance if it occurs at the margin of Shrapnell's membrane, and is then apt to be associated with processes in the vault and antrum which discourage one from expecting much from local treatment.

(*c*) Marginal perforations are usually associated with severe processes. There are apt to be extensive granulations, necrosis of bone, or cholesteatoma. Besides the small marginal perforations (Fig. 80) the large perforations often derive a certain amount of significance from the fact that they encroach upon the tympanic ring, and may on this account be alluded to as marginal perforations. An opening in the membrana tympani which is located near the tympanic ring may, at times, easily be

mistaken for a marginal one. Therefore one must be absolutely certain that no drum membrane exists between the opening and the ring. Marginal perforations as a rule offer very little prospect of cure by local (non-operative) treatment.

(*d*) Perforations in the posterosuperior quadrant are as a rule associated with involvement of the vault and antrum, the path of drainage being along the long process of the incus. When the discharge has been continuous and possesses odor, these perforations usually indicate the necessity of a radical operation.

(*e*) Multiple perforations, while not pathognomonic of tuberculosis of the middle ear, usually only occur in

FIG. 80.—Small marginal perforation. The patient whose drum membrane is shown had a continuous odorous discharge for years. Operation revealed the tympanum and antrum full of granulations.

this disease, and should therefore, when present, excite suspicion that the process is of this nature.

Perforations Involving Shrapnell's Membrane.—Perforations situated either wholly or partly in Shrapnell's membrane, while not as common as those in the other parts of the membrana tympani, are of great importance and may easily be overlooked unless one forms the habit of carefully inspecting this part of the fundus in every patient. In many of these perforations the secretion is scanty, frequently not sufficient being discharged to appear at the external end of the meatus, but it dries in crusts near the perforation in the depth of the canal. Many perforations may not be seen if these crusts, which

either cover the opening or granulations concealing it, are not removed. When patients come complaining of deafness, tinnitus or a full feeling in the ears and crusts are discovered, a perforation will not infrequently be found to be present, although the history of a discharge is absent. Perforations in Shrapnell's membrane may be divided into two general classes: (*a*) those which involve the membrana tensa and extend into Shrapnell's; (*b*) those which involve Shrapnell's membrane but do not extend into the membrana tensa.

(*a*) In the former the defect in the membrana tensa may be either the large or small type. If large, the usual location is in the posterior part of the membrane, the

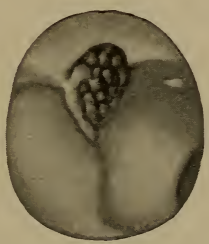

FIG. 81.—Large posterior perforation extending into Shrapnell's. The head of the malleus is exposed. In this patient the radical operation showed a large antrum full of granulations. The discharge possessing odor had been continuous for many years.

defect in Shrapnell's being near the malleus, as in Fig. 81, or more posteriorly near the margin of the Rivinian segment. The internal tympanic wall may be dermatized and show more or less distinctly the anatomical arrangement, as in Fig. 81; or it may be covered with thickened mucous membrane in any stage of granulation or polypoid formation. Very rarely these perforations extend into Shrapnell's anterior to the manubrium. They have the same significance in either case. The discharge is apt to be freer than in perforations limited to the membrana flaccida. They are as a rule associated with granulations, necrosis of bone or cholesteatoma in the vault

and antrum—processes which require operation for their cure.

If the defect in Shrapnell's is a part of a small perforation, the location may be either on the posterior or anterior folds, the former being oftener seen. Sufficient of the membrana flaccida is destroyed to open the vault, and it is from this location that the pus comes. The processes which they accompany are the same as in those limited to Shrapnell's and usually require operation for their cure.

(*b*) Of the perforations in Shrapnell's many are on the margin of the Rivinian segment and are more apt to con-

Fig. 82.—Perforation (filled with granulations) on the margin of Shrapnell's. The patient had scanty odorous discharge for years. Operation showed necrosis of the walls of the vault; cholesteatoma and granulations filling the vault and antrum. The membrana tensa was practically normal.

tain granulation tissue than those more centrally located. The membrana tensa may be practically normal, as in the patient whose membrane is shown in Fig. 82; or more or less cicatricial, showing that the process has exhausted itself in the atrium but continued in the vault and antrum. These perforations may extend into the dermal layer of the superior canal wall. A lesion which while perhaps not anatomically of this class yet clinically is so closely related that it belongs here, is a perforation through the integument on the canal wall adjacent to the membrana flaccida. The purulent secretion, dissecting its way out beneath the periosteum, ruptures through the canal wall or extension of necrosis of bone from the margin of the

vault may be responsible for their formation. They are usually filled with granulations, but a small probe may be passed into the vault. Both of these types of perforations are apt to be associated with cholesteatoma, caries of the walls, and granulations in the vault and antrum. In many of them the antrum seems to be enlarged, and this outside of the changes due to cholesteatoma. They indicate operation.

Central perforations of Shrapnell's may be of sufficient size to expose the head of the malleus or very much smaller, their presence being revealed only upon close inspection. At times, granulations are evident in the depth of the perforation, on its margins or pouting into the canal, completely filling the opening in Shrapnell's. The secretion is usually scanty and of foul odor, drying into crusts. Usually, an operation is required for the accompanying disease in the vault and antrum. Patients with healed perforations in Shrapnell's have been seen by Politzer. At times one feels that a condition producing such a small perforation and so scanty a discharge cannot be of a very serious nature; but this impression is not justifiable. The author has yet to see an operation performed for a process with a perforation in Shrapnell's, in which the findings did not justify the procedure.

Perforation in Prussak's Space.—One form of perforation in Shrapnell's occasionally seen is due to an exhausted suppuration in Prussak's space. The perforation is dry and merely the result of an inflammation limited to this space, in which its external wall has been destroyed. The neck of the malleus forms the bottom of the depression. It is really a residual process and is mentioned in this place that it may not be confused with real perforations in Shrapnell's which are of serious import.

Complications.—A chronic suppurative process may at any time develop a complication. The aural pus from chronic suppurating ears usually contains epithelial masses, round cells, saphrophytic microörganisms and a variety of bacilli, but during an acute exacerbation some

of the microörganisms associated with acute processes are usually present, such as streptococcus, pneumococcus, etc. This justifies the belief that the cause of an acute exacerbation is an acute infection engrafted upon the chronic process. The discharge usually increases in amount and becomes frankly purulent. In these acute exacerbations the process acts very much as an acute purulent otitis media. There is pain and fever and the mastoid very frequently becomes involved. The condition is properly considered as being a serious one on account of the danger of intracranial complications or labyrinthitis, which are more common with acute exacerbations of a chronic process than with the simple acute otitis media. When the mastoid becomes involved under these circumstances, no time should be lost in waiting to see if it will not resolve, but the operation should be done at once. By this means further complications may, if possible, be prevented. The procedure demanded in these patients is usually the radical operation, although under exceptional circumstances one may be justified in first performing the simple mastoid and then later the radical if necessary, which it usually will be. The other complications of chronic suppuration are labyrinthitis, meningitis, sinus thrombosis and brain abscess. They are discussed in separate chapters.

Diagnosis.—The diagnosis as a rule is not difficult. That the patient has an O. M. P. C. is shown by the presence of a discharge which has existed for over three months, and the loss of integrity of the drum membrane, or, in rare instances, a perforation on the canal wall giving exit to the pus. If one is not careful one may fail to make a diagnosis in those patients with scanty discharge, drying in crusts upon the canal wall. These types are described under Perforations Involving Shrapnell's Membrane. An attempt has been made to show what, more than the simple fact that the patient has O. M. P. C., may be learned from the study of the fundus appearance. In making out cholesteatomatous processes, if one is

able to remove some of the material from the middle ear, its appearance and odor are characteristic. Microscopic examination shows the presence of cholesterin crystals, epithelial débris, perhaps also the nests. Cholesterin crystals may be found in epithelial masses coming from the meatus, but the nests or the fact that the mass submitted to examination was taken from the middle ear establishes the nature of the process.

Prognosis.—The prognosis is serious in processes involving the vault and antrum, less so in those patients in whom these parts are not involved and the discharge is intermittent. While many patients with the former type may live for years, still the disease is more of a menace to life and health than is generally supposed. Pathologists performing autopsies in the large city morgues state that the cause of death is some complication of chronic purulent otitis media, in a much larger proportion of cases than is generally supposed. From the description of the disease as given above, one should be able, insofar as it is possible, to separate those processes which are a menace to life and health from those which are more benign in their effects. In a great many patients the disease can be cured either by local treatment or by operation, still the prognosis as given to the patient should be a guarded one.

Treatment.—When one is able to find and remove the cause of the suppuration, it is the first step in treating the patient. When adenoids are responsible, much may be expected from their removal, especially if the middle-ear process is mainly confined to the atrium and tube. They should be thoroughly removed and the tonsils enucleated at the same time if they are enlarged or small and infected. Any other abnormal condition of the nose, nasopharynx or accessory sinuses may be attended to, but not much improvement of the aural condition may be expected to be produced by doing so—at least not in comparison with that which often follows the removal of the adenoids.

The general health is to be attended to in order to

increase the resistance. With this end in view, vaccine therapy has been tried, and while much was expected from it, the results have been disappointing. Benefit has been reported in some instances, but the general opinion of aurists is that it is without marked value. The author's experience does not encourage him to expect much from this mode of treatment. There are so many germs in a chronic discharge that the difficulty of growing the one, if there is one, which is responsible for the disease is very great. Perhaps bacteriologists will eventually overcome the difficulties and furnish the aurist with a remedy of value.

In treating the disease great attention must be given to cleanliness. The discharge must be thoroughly removed. The best way to do this is by syringing with antiseptic solutions. Bichloride of mercury, 1 to 4000, or a saturated solution of boracic acid, seem to fulfil the requirements. The frequency of syringing depends upon the amount of discharge; usually four times a day is sufficient. If there are pockets of pus that this fails to reach, peroxide of hydrogen, the ten-volume solution diluted one-half, may be instilled ten minutes before syringing the ear. There seems to be no danger in using this peroxide solution. In large perforations good results are often obtained by the instillation of alcohol. A 1 to 2000 bichloride solution in 50 per cent. alcohol is used and ten drops are instilled twice a day, the patient holding the head well to the opposite side, that the solution may come in contact with the intratympanic structures to as great a degree as possible. After the patient is accustomed to the use of this strength of alcohol, it may gradually be increased until 95 per cent. alcohol is used or pain is produced. When there is reason to suppose that the Eustachian tube is diseased, inflation followed by the application of nitrate of silver or argyrol solution to the tubal mucosa may produce good results. (For method see Tubal Catarrh.) An attic syringe (Fig. 83) may be used to cleanse the vault and antrum. This is

a small syringe with a curved point which is inserted through the perforation and directed upward. Dilute alcohol is used in this manner, as it more readily brings away the cholesteatomatous masses than water, which may cause them to swell. Granulations, if within reach, may be cauterized with a fused bead of silver nitrate or removed with the sharp ring curette.

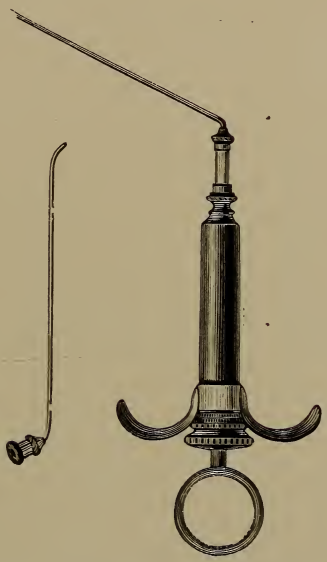

Fig. 83.—Blake's attic syringe.

Dry Treatment.—Sometimes the insufflation of boric acid is of value, especially if large perforations are present. The treatment, at one time in vogue, of filling the canal with powder is to be condemned, as by absorbing the secretion it may harden and close the perforation or canal and thus interfere with drainage and produce dangerous complications.

Removal of Polypus.—If a polypus is present, it should be removed. In many instances polypi are of sufficient size to require removal before an examination of the

fundus of the meatus is possible. The large variety which project well toward the entrance of the meatus are usually best taken with the snare. One with as small a cannula as will carry a stiff wire is most conveniently used. The loop is placed over the growth and gently pressed well to the fundus of the canal. Gentle traction on the growth while the loop is being passed will prevent its folding upon itself in front of the loop, which will make it difficult to reach the fundus with this part of the instrument. All of the manipulation should be very gently performed and, on account of its painful nature, it is frequently advisable to administer gas. The loop, being in position, is now drawn home and the growth severed without exerting any traction. The canal may now be packed with cotton saturated with adrenalin to stop the hemorrhage. This being accomplished, the base of the polyp

FIG. 84.—Sharp ring curettes.

is examined to ascertain if sufficient has been taken. For removal of this base, or for a somewhat sessile polypi, or as a matter of preference in any form, the sharp ring curette may be used (Fig. 84). It is usually necessary to use one with a large ring and a narrow band so that the fenestra may be as large as possible. After determining the attachment of the growth, the ring is worked over the polyp and pressed as near to the location of the attachment as possible and this is severed by forcing the cutting edge of the curette through it. A polyp should never be torn away, as no one can foreknow the exact location to which it is attached. It may spring from the tegmen tympani and its evulsion may result in injury through which infection may reach the meninges. A number of deaths have been caused in this way. After removal of a polyp the patient should be kept under close observation for twenty-four to forty-eight hours,

so that should complications arise they may be promptly treated.

Indications for Operation.—After persevering in the treatment of an O. M. P. C. for a time without improvement, the question of operation arises. An attempt has been made, under fundus appearances, to show in what processes one may hope for a cure by other means. In addition to these, the discharge affords information. Some patients with a discharge of foul odor, which has existed for years, with fundus changes indicating that the pus comes from the vault or antrum, should be operated without preliminary treatment. Others, who do not have these fundus changes, in whom the discharge has not existed so long, and especially if they give a history of periods of cessation of the discharge, are entitled to a faithful attempt to secure a cure without operation. No fixed time can be given either for duration of the discharge or continuance of treatment before an operation becomes advisable. When one feels certain that local treatment is a failure, and that the process is of such a nature that the health is menaced or complications are threatened, it is time to operate. Cholesteatomatous processes practically always demand operation.

Form of Operation.—Before deciding upon the radical operation, it would seem wise to consider whether or not minor surgical procedures might not result in a cure. Several measures have been advocated, some of which are still on probation, while others have given place to procedures more certain of results. They may be divided into three classes:

1. Those which are intended to produce closure of the Eustachian tube.

2. Those intended to remove necrotic bone (mainly the ossicles) through the external auditory meatus.

3. A modified radical operation which for the sake of producing less impairment of hearing, while opening the antrum, leaves undisturbed the ossicles and drum membrane.

Tubal Closure.—In those patients whose middle-ear suppuration continues on account of some condition existing in the Eustachian tube, there can be no doubt but that permanent closure of the tube is desirable. Yankauer[1] has devised an operation which is intended to produce closure of the osseous portion of the tube. By means of a specially designed curette he removes the mucous membrane lining the tube from the tympanum to, and including, the isthmus. If the operation is successful, this portion of the tube becomes filled with cicatricial tissue. While too much has been expected of this operation, and it has, no doubt, been performed in many unsuitable cases, still it promises to be a procedure of value. The type of processes in which it should be of value are those in which local treatment usually improves the condition and it remains more or less quiescent until lighted up by a fresh infection, occurring through the Eustachian tube. It is difficult to see how closure of the tube would be of any value when the vault and antrum are filled with granulations or cholesteatomatous masses and their walls the seat of bone necrosis or caries, many of which processes persist long after nature has permanently closed the tube at its tympanic orifice.

Yankauer's Operation.—The operation is performed by anesthetizing the tube and middle ear, after which, if necessary to gain access to the tube, a flap of drum membrane is removed. The mucous membrane is then curetted from the osseous portion of the tube by Yankauer's curette, introduced through the meatus. Care should be taken not to use much force in an inward direction, as the carotid artery is in the immediate proximity, and while the artery has not so far been wounded, the bone has been removed from it in a patient coming under the author's observation.

Other Methods of Tubal Closure.—Mosher has attempted to close the tube from the pharyngeal end by applications

[1] Laryngoscope, July, 1910.

of silver nitrate in strong solution. The subject of tubal closure is one that is attracting the attention of aural surgeons, and progress in this line may reasonably be expected. As a preliminary to the radical operation and subsequent to it in those patients who are not completely relieved of their discharge on account of the tube remaining open, some method or form of treatment which will produce permanent closure of the tube is desirable.

OSSICULECTOMY.

The second class of these operations is the removal of the malleus and incus with the remains of the tympanic membrane. This procedure may at times be adopted with success.

Indications.—It is indicated in those processes which are due to necrosis of the ossicles and when it is believed that the increased drainage of the vault and antrum afforded by their removal may result in cure of the process. It is at times possible to make out necrosis of the manubrium by inspection. Necrosis of the incus may be suspected if a large posterior defect in the membrana tympani enables one to see the stapes in position with the long process of the incus absent. Unfortunately, one cannot be sure that the remains of the incus are still present or that they have any influence on the suppurative process. Perforations in Shrapnell's may exist through which necrosis of the ossicles may be made out. However, when these conditions are present, as well as in patients in whom they are absent, the involvement of the ossicles may be such a small part of the process that their removal does not have much influence for good. As compared with the radical operation, by means of which every part of the tympanum and antrum is exposed and becomes accessible, ossiculectomy is a weak procedure. As for removing granulations from the vault, as advised by many advocates of the operation, it is not free from danger. So the operation when performed

should be limited to the removal of the malleus and incus with the remains of the drum membrane, and curetting any granulations which may exist in the lower part of the tympanic cavity.

Anesthesia.—The middle ear is thoroughly douched with bichloride solution before operation. Children and nervous subjects require general anesthesia. The operation, however, can be made practically painless by local anesthesia which is induced as follows: A few minims of a 10 per cent. solution of cocaine is instilled into the middle ear through the perforation and allowed to remain ten minutes. If the defect in the drum membrane is a large one, a pledget of cotton saturated with the solution is placed in contact with the internal tympanic wall. This aids in making the operation painless by producing anesthesia of the mucous membrane with which the solution comes in contact, but the main reliance is placed upon the hypodermic injection of one-half of 1 per cent. solution of cocaine to which two or three drops of adrenalin have been added to each cubic centimeter. The needle is entered upon the superior wall of the canal at the juncture of the osseous and cartilaginous meatus and a few drops forced out at a time as the needle travels along the bone to the internal end of the osseous meatus. Anesthesia becomes complete in about ten minutes.

Operation.—The instruments advised are Politzer's ossiculectomy set (Fig. 85). The largest speculum possible is now inserted and the operation performed under brilliant illumination. The tympanic membrane is completely separated from the tympanic ring by a small scalpel. It is well to start this with a knife having a very sharp point, then to substitute one with a probe point in order not to wound the mucous membrane, and thus increase the hemorrhage.

The next step is dividing the incudostapedial articulation. This will not be necessary if there has been necrosis and sloughing of the long process of the incus. This is accomplished with a small knife, the blade of which is

at a right angle with the shaft. This is inserted from in front between the long process of the incus and the internal tympanic wall with which the flat of the blade is in contact. Upon rotation of the instrument, the blade moves downward and severs the connection between the incus and stapes. If there is an adhesion between the manubrium and the internal tympanic wall, it may be divided in the same manner; or a ring knife, resembling

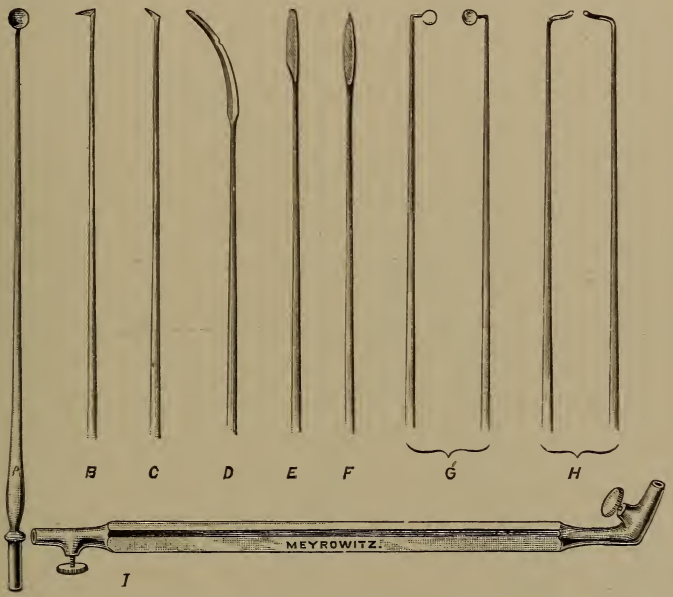

FIG. 85.—The Politzer set of instruments for performing ossiculectomy.

the sharp ring curette, may be slipped over the end of the manubrium with the cutting edge upward, and gradually forced through the adhesion, or if there is no adhesion, this instrument gradually worked upward will sever the tendon of the tensor tympani and the ligaments holding the malleus in position. This loosens the ossicle and it may be delivered with a small angular forceps.

The incus is now removed with a hook designed for

that purpose. These hooks are made right and left. Selecting the appropriate one, it is worked up posterior to the ossicle, then by rotating the instrument, the hook with the incus engaged is brought down into the atrium and removed with the forceps. Considerable skill is at

Fig. 86.—Dench ear punch.

times necessary to remove this ossicle, as it may be forced into the antrum or lie concealed internal to the tympanic ring. If it escapes into the former location, it cannot be removed; if in the latter, a careful search will enable the surgeon to find and remove it. Care should be taken

when inserting the hook not to disturb the stapes or structures in the oval window, and no traumatism should be inflicted upon the Fallopian canal or facial paralysis may result.

The granulations in the atrium may be removed with curettes, taking care not to wound the facial nerve. An attempt may also be made to close the orifice of the Eustachian tube by curetting the mucous membrane at its tympanic orifice. If the internal end of the superior canal wall is prominent, it may be removed with a properly designed biting forceps (Fig. 86). By thus taking away the prominent margin of the Rivinian segment much better drainage of the vault and antrum is secured. As stated above, it cannot be considered a safe procedure to attempt to do much curetting in the tympanic vault. It is well at the outset to acknowledge the limitations of this procedure and not try to accomplish with it that which can only with safety be accomplished by the radical operation.

After-treatment.—Having completed the operation, the ear is syringed with normal saline or bichloride solution and a strand of iodoformized gauze lightly packed into the tympanum. Sterile gauze, cotton and a bandage complete the dressing. This may be changed daily for a few days, after which the ear is left open and regularly irrigated.

Accidents.—Accidents which have occurred are as follows: facial paralysis from wounding the nerve as it lies in the internal tympanic wall; injury to the jugular bulb either in attempting to remove necrotic areas from the tympanic floor or in curetting in this location when a dehiscence was present; meningitis from injury to the structures in relation to the tegmen tympani, produced while attempting to remove granulations from the vault. The chorda tympani is always divided and permanent loss of taste sense results on the anterior two-thirds of the tongue. The patient is not usually conscious of this fact, and as it is present in about 50 per cent. before the operation, it cannot be considered of importance.

Results.—Some patients are cured of the discharge. The proportion is, however, small as compared with the radical operation which frequently is required later. Some are benefited to a greater or less extent, while a a considerable proportion show no improvement. This uncertainty of result, and the feeling which the surgeon has that he cannot be certain that he is removing all of the diseased tissues, have been responsible for the lack of popularity of this operation.

Modification for Non-suppurative Cases.—The operation when performed in a non-suppurative ear to increase the hearing is in the main as described above, with this exception, that care is taken not to wound the mucous membrane except to divide adhesions in the niche of the oval and round windows. The ring knife is not used, as it inflicts too much traumatism. The tensor tympani is divided with the angular knife and the ossicles carefully delivered. In the after-treatment irrigations are not used unless absolutely necessary on account of infection. If the drum membrane reforms it must be removed a second time, as the success of the procedure rests upon having it absent.

Modified Radical.—The third class of operations is discussed under the head of Modified Radical Operation after the Radical Operation.

THE RADICAL MASTOID OPERATION.

Instruments.—The instruments needed for the performance of the radical operation are the same as for the simple mastoid, and the following in addition:

1. Two Richard curettes (Fig. 87), small and medium-sized. These are bent in the shank and are very useful for scraping away hard bone.
2. One small chisel.
3. One small curette for use in the tympanic opening of the Eustachian tube.
4. One sharp ring curette with flexible shaft.

5. One long narrow-bladed knife for cutting the meatal flap.

Preparation of Patient.—The preparation of the patient is in general the same as for the simple mastoid operation. As the radical operation is not so frequently an emergency procedure, usually sufficient time is afforded to thoroughly prepare the patient. He should be admitted to the hospital the day before and a thorough test of the aural functions made. The amount of hearing, the upper- and lower-tone limits and the tuning-fork tests for bone conduction affords a basis for judging of the condition of the acoustic labyrinth, and also gives data for comparison as to the results of the operation. The rotation, caloric and fistula tests should be made as having an important bearing on the kind of operative

Fig. 87.—Richard's curette.

procedure adopted, and also in studying any labyrinthine complications or symptoms which may arise later (see Suppurative Disease of Labyrinth). If it is proposed to insert a Thiersch graft, the thigh should be prepared the night before if possible. The left is usually selected as being slightly more convenient than the right. It is scrubbed with soap and water, shaved, washed with alcohol and done up in antiseptic dressings.

The patient should have a general anesthetic. The position of the assistants and surgeon is the same as in the simple mastoid operation.

Incision.—The incision varies slightly from that in simple mastoidectomy. It can be started a trifle higher than the apex of the tip and does not go through the muscle to the bone at its lower end. As it is extended upward, it is made a trifle more posteriorly and its upper

end is carried slightly more forward. It extends to the bone in the middle of its course and through the temporal fascia where it lies over that muscle. It is made more posteriorly in order that as the flaps are sutured in completing the operation the line of junction may overlie the bone and not the radical cavity. This facilitates healing and enables one to place the grafts properly. The incision is extended farther forward above the ear, as it is necessary in this operation to have a much greater exposure of the upper wall of the canal and the ear must be drawn farther forward to afford this exposure.

Exposing the Field.—After completing the incision all of the bleeding-points are caught with hemostat forceps. The anterior flap is pushed forward with the periosteal elevator, exposing the posterior and superior margins of the meatus and the anterior retractor inserted. The fibers of the temporal muscle are elevated in an upward and forward direction. The ear can usually be drawn sufficiently forward with incision of the temporal fascia, but if necessary to expose the upper margin of the bony meatus the muscle also may be freely incised. Unless the fibers of the sternomastoid are attached unusually high on the surface of the mastoid, they need not be elevated. The skin and periosteum is now separated from the external auditory canal over the superior and posterior walls, but the anterior wall should be undisturbed. The posterior margin of the incision may be elevated at any time if more room is required.

Opening of the Antrum.—One who has been accustomed to performing the simple mastoid, must not be surprised if he does not encounter cells, as the bones in processes requiring the radical operation are apt to be of the sclerotic or infantile type. In the latter type the sinus may be well forward and the middle fossa low. There are no constant landmarks on the bone which indicate the position of these structures. A low temporal ridge or a shelving upper canal wall are very apt, however, to be associated with a low middle fossa. The Dench triangle

is located (Fig. 69). This overlies the antrum which will be opened at a depth of three-eighths to one-half inch, varying to a certain extent with the size of the antrum. These measurements are from dry bones. One seems at times to go deeper than this before opening the antrum. If the posterior wall of the canal extends forward as it approaches the drum membrane, the antrum will be forward since it lies contiguous to the junction of the superior and posterior walls of the meatus very near the insertion of the tympanic membrane. So in working in a bone with a shelving posterior wall, as above described, one must work forward. To use the gouge and excavate perpendicular to the surface of the mastoid with such a configuration of the parts would endanger the sinus if it is located far forward. Frequently, the antrum is large and offers no difficulty. But a small antrum, deeply placed and especially with a shelving posterior canal wall, is certain to be trying to a surgeon of limited experience in this operation. A large-sized gouge is used and a shaving is taken, starting upon the surface of the mastoid, very near the margin of the meatus. The upper edge of the shaving is in the triangle and the lower is toward the floor of the canal. The gouge is very nearly parallel with the posterior wall of the canal, and the first shaving is largely composed of the canal wall. By taking repeated shavings in this manner, one works a gutter in the posterior canal wall, about one-fourth inch in length, that is, measured from the mastoid surface toward the drum. In working backward each shaving is carefully taken, as the sinus may be exposed at any time when, of course, further progress in this direction is impossible. If the sinus should not be so far forward, one deepens the gutter backward only enough to afford sufficient room to excavate a pit in its internal end, which naturally is toward the antrum. By forming this gutter the triangle has been moved from the surface of the mastoid, inward the length of the gutter, and it is therefore that much nearer the antrum. It is in the offset which forms the

inner end of the gutter and in the triangle adjacent to the union of the superior and posterior walls that a pit is now excavated, with a small gouge, to open the antrum. It may be that this cavity or soft bone overlying it has already been encountered; if so, the opening is enlarged with the curette. If not, the pit is deepened, working parallel with but not taking down the canal wall, until the antrum is opened. One must keep in the triangle

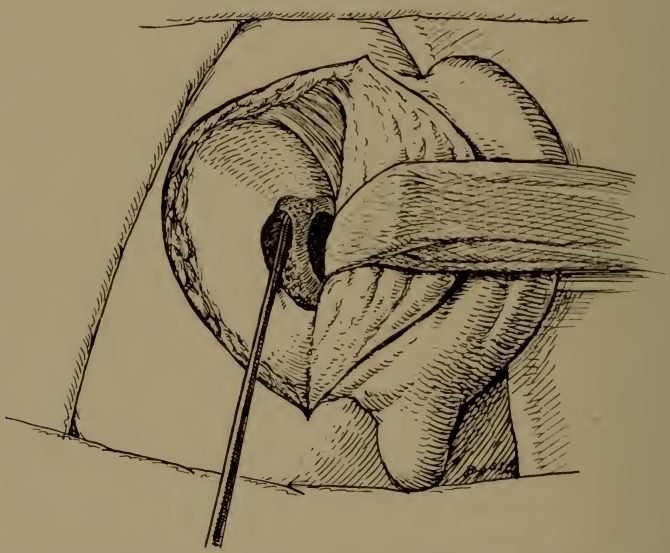

Fig. 88.—Radical operation. Antrum opened with curved probe inserted.

in deepening the pit. If one works too high, the middle fossa will be opened; if too low, the antrum will be missed and the facial nerve endangered if the excavation is carried deeply enough. Fortunately, the very small antra which require this great care are not the rule. That the antrum has been opened may be known from the fact that a bent probe readily passes forward into the vault (Fig. 88). After the antrum is opened, or before, if difficulty is experienced in this step of the opera-

tion, a gauze retractor is inserted in the membranous meatus. This is simply a strip of gauze which is passed into the depth of that part of the auditory meatus which is still attached to the auricle, starting from its anterior aspect, drawn out behind, and brought forward over the posterior surface of the auricle (see Fig. 88). The assistant makes gentle traction on this, thus exposing the depth of the osseous meatus.

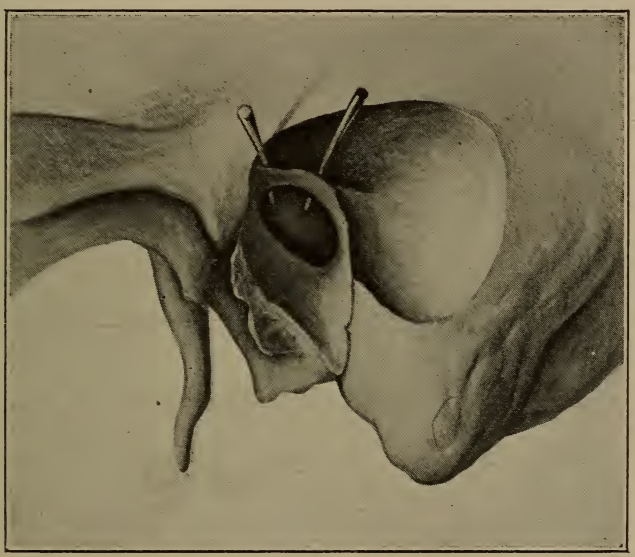

FIG. 89.—Radical operation. The figure illustrates the first step in taking down the bridge.

Forming and Taking Down the Bridge.—The author has found this step of the operation a difficult one for the student to understand from descriptions alone, but once performed upon the cadaver it becomes very plain. The upper and anterior part of the mastoid opening is now extended forward toward the zygoma. The distance to which this can be done depends altogether on the thickness of the superior wall of the canal. If there are

cells or diploetic tissue between the cortex of this wall and the inner table (as in the specimen from which Fig. 89 was made), the antral opening can be made to extend well forward even to the line of the anterior canal wall. This opens the vault of the tympanum and the bridge of bone between the canal and the upper cavity occupies an anteroposterior position. If, on the contrary, the floor of the middle fossa dips down, so that the cortex of the superior canal wall and the inner table exist in the same hard bone, and this of no great thickness, the bridge will be more or less vertical. The best instrument to remove this soft tissue between the cortex of the superior wall and the inner table is a small curette. It is inserted into the antrum (or vault as one works forward) and a small hold taken with the hollow of the bowl directed outward and forward, and by using the posterior margin of the bone wound as a fulcrum, made to travel outward; at the same time it is gradually rotated so that the back of the spoon is directed more and more toward the inner table, thereby preventing this structure from being removed and the dura thus exposed. As this work progresses the outer end of the superior canal wall is taken down.

The small gouge is used anteriorly and is directed parallel with the anterior canal wall and small shavings removed, working forward, thus lengthening the bridge, while with a small chisel shavings of bone are taken from behind forward to meet the cut made with the gouge. It will be noticed that in using the chisel in this way it points toward the dura. It must therefore be under good control and be gently driven by the mallet and only to the cut in the bone already made by the gouge. Fig. 89 shows the cells and diploe removed and part of the superior canal wall taken down. It also shows why this partition between the two cavities is called a bridge. The bristles are passed from the vault into the atrium. This septa of bone forms an arch over the vault, its internal surface being a part of the outer wall of the vault. It is

therefore a bridge, the posterior abutment of which is that part of the posterior tympanic wall adjacent to the additus ad antrum, while its anterior abutment is part of the anterior tympanic wall (superior wall if the bridge from anatomical conditions must be formed vertically). A curved probe passed from the antrum or upper cavity into the atrium, and pressed as far backward as possible against the posterior tympanic wall, lies upon the Fal-

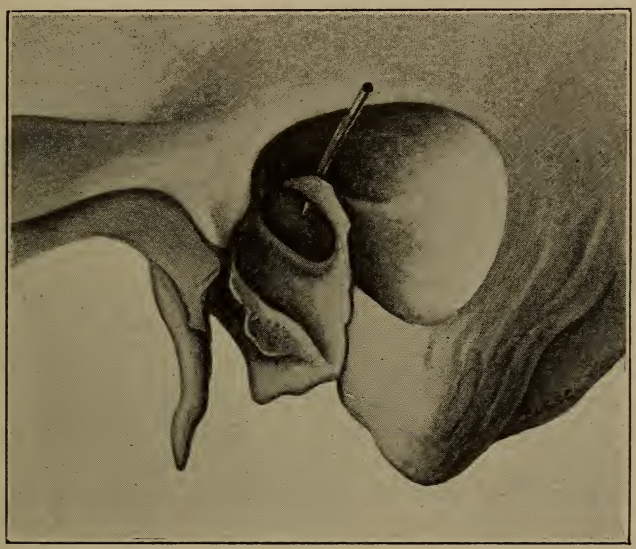

Fig. 90.—Radical operation. The bridge taken down, showing well-marked posterior overhang.

lopian canal at about the junction of its tympanic with its third or descending part. This canal containing the facial nerve lies directly internal to the bend in the probe. Immediately above the facial lies the anterior half of the horizontal semicircular canal, which is now in plain view. On account of the nerve, in taking down the bridge it is desirable to work as far forward as possible. By using the gouge and chisel as above described, and

taking a thin shaving at a time a V-shaped piece is removed from the bridge, the anterior arm of the V being as nearly level with and in the same direction as the anterior canal wall as possible, while the posterior arm slopes gradually across the bridge to meet the anterior arm at the apex of the V which is directed toward the tympanum. In taking out the last shavings, one must exercise great care that the instrument does not go through the bridge and impinge upon the internal tympanic wall, where it might do great damage. In all except the hardest bones, the curette may be used to complete this division of the bridge. It is inserted as in extending the antrum forward and rotated so that the cutting edge is in contact with the bridge well forward, then with increased pressure with its anterior lip it is withdrawn, at the same time scraping the upper surface of the bridge, that is, the surface toward the middle fossa. If, while pressing this instrument against the bridge, it goes through, no harm will be done. Thus the bridge is divided in such a manner as to leave a well-marked posterior overhang (Fig. 90).

Removing the Overhang.—If there is an anterior overhang it may be removed with a curette or small gouge. If the posterior overhang is quite long and there is no room to use the small curette between its point and the anterior wall, it may be shortened by chiseling off a few fine shavings. The remainder can be safely removed with the small curette, which is inserted internal to it with one cutting edge pressed against it (Fig. 91). By using the upper border of the bone wound as a fulcrum, the spoon of the curette is moved directly outward in the line of the external auditory canal. Only a little should be taken at a time and the probe should be used each time to determine the amount of overhang still remaining before an additional piece is taken. In removing the last piece, the side of the curette is in contact with the bend in the Fallopian canal, and great pains should be taken not to allow the curette to rotate and thus produce pressure with the back or side of the bowl

against this canal, as a depressed fracture of the Fallopian canal wall would be produced and facial paralysis result. In this work so close to the nerve, the anesthetist watches the face carefully and should any twitches be seen, imme-

FIG. 91.—Method of using the curette in taking down the posterior overhang.

diately reports the fact to the surgeon, so that having warning of the maneuver which disturbed the facial nerve and thus produced the twitches, he may not repeat it. The object in taking down the posterior overhang flush with the posterior tympanic wall is that if it is not

done a cavity will be left which interferes with subsequent healing or the taking of grafts if they are used. While removing the posterior overhang a dry field is necessary; therefore, as soon as there is sufficient exposure of the tympanum, the ossicles are taken out and granulations removed with a sharp ring curette, with flexible shaft, as from the latter troublesome bleeding often takes place. The wound should be packed with gauze saturated with adrenalin solution to arrest the hemorrhage and obtain a dry field.

Lowering the Facial Ridge.—The ridge of bone which will now exist, partially dividing the cavity formed while opening the antrum from what remains of the osseous external auditory meatus, and known as the facial ridge or spur, is now to be taken down as far as possible with safety. For this purpose, the Richard curette is undoubtedly the best and safest instrument, although a part of it may with safety be removed with the chisel. In lowering it, an attempt is made to enlarge the size of the opening leading to the tympanum. This is accomplished by scraping the facial spur on its external and anterior aspect. The horizontal semicircular canal is in plain view and is the guide to the position of the nerve. A line dropped from this canal directly downward, that is, parallel with the median line of the body, is supposed to be just without the facial nerve and theoretically one may remove this mass of bone to this line. In practice, however, one stops somewhat short of this, as instances are on record in which the nerve has occupied a position somewhat external to this line. The point at which the nerve is endangered, if it is at all, is near the semicircular canal.

Lowering the Floor.—In some subjects the inferior wall of the external auditory meatus is high and its inner end, surmounted by the tympanic ring, causes a cavity to exist in the middle ear, the hypotympanum or cellar. If this condition is present, the floor must be lowered and the arc of the tympanic ring removed, so that the inferior

wall or floor will be made flush with the floor of the canal. The tympanic ring may be taken with the curette which is inserted internal to it and moved outward, thus scraping this structure flush with the tympanum. The position of the jugular bulb beneath the floor of the middle ear, and in proximity to the end of the curette while doing this work, must be borne in mind. For lowering the inferior canal wall the gouge is used and shavings removed until it is sufficiently low.

Eustachian Tube.—A small curette is inserted into the tympanic orifice of the Eustachian tube and the mucous membrane removed if possible to the isthmus. The carotid artery lies internal and adjacent to the tube, therefore all manipulations in this region should be made carefully. Insofar as possible, the cutting edges of the curette should be directed outward and forward. The tympanic ring at the tubal margin may also be taken down.

Cleaning the Cavity.—Three important structures are to be borne in mind while cleaning the cavity. These are the facial nerve, the oval window with the stapes in position and the round window covered in by a thin membrane. If a sharp ring curette with flexible shaft is used to remove the granulations, mucous membrane and débris, these structures will be safeguarded as far as it is possible to do so. Such an instrument glides over the Fallopian canal when a stiff curette of different shape would remove part of the wall, perhaps injure the nerve also. Very little curetting should be done in the region of the oval and round windows. With the ring curette the stapes, if in normal position, will escape injury. The author has never disturbed it in this manner but once. In this patient its foot-plate was necrosed and the ossicle lay in a mass of granulations (Fig. 78). The patient made a complete recovery, without untoward symptoms and with increased hearing. In situations remote from these structures a Spratt curette may be used to remove granulations, mucous membrane and carious areas of

bone. The best operators devote much pains to this thorough cleaning of the cavity and much of the success of the operation depends upon its being done carefully and well. This is especially true if a primary skin graft is to be inserted.

Meatal Flap.—The depth of the cavity is packed with gauze, which is saturated with adrenalin if it is to be grafted. The bleeding-points are tied and the hemostats removed. The gauze retractor is taken out and the surgeon proceeds to form what is known as the meatal flap. The parts from which this is formed are the concha and the skin which lined the external auditory canal and which was separated from this early in the operation. These parts constitute more or less of a membranous tube lined with the dermal epithelium. The object in cutting this flap is to open this tube and spread it out on the walls of the radical cavity so that dermatization may begin from its epithelial margins, and at the same time to make a larger opening in the auricle through which the cavity can be taken care of during the healing process.

A variety of methods have been devised for accomplishing this purpose, some of which will be briefly noticed later. The flap advised as answering every purpose is the one devised by Ballance and slightly modified by Dr. Dench. Ballance splits the meatal tube through the middle of its posterior aspect and reflects the flaps in either direction, while Dench splits the tube along the lower part of its posterior aspect thereby having but one flap to reflect and that upward. The conchal incision is about the same in either method. It is this flap as thus modified that will be described. The narrow-bladed knife is inserted into the opening in the auricle corresponding to the external auditory meatus and passed through the tube until it emerges at its inner end, which is now posterior as the auricle is drawn forward, the flat of the blade being pressed against the inferior wall with the cutting edge backward. With a sawing motion, but with very slight pressure in the direction of the cutting edge,

the tube is cut through until the blade enters the concha. This part of the procedure takes place with the operator watching the posterior aspect of the tube the greater

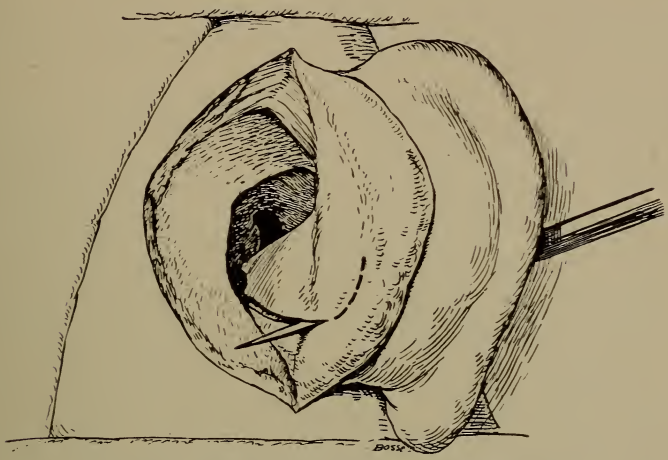

FIG. 92.—First step in cutting the meatal flap.

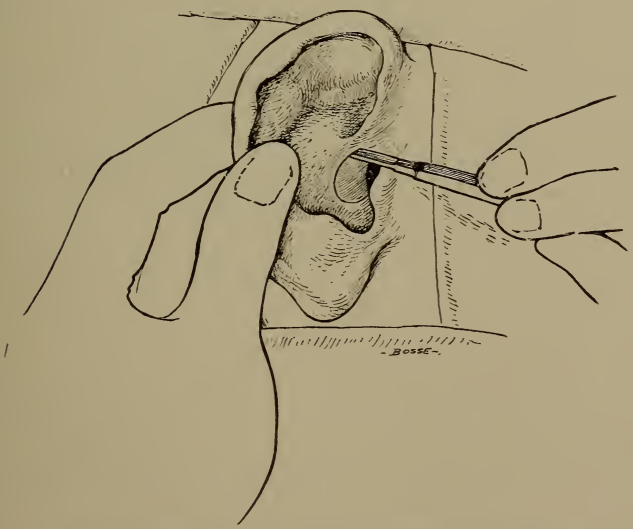

FIG. 93.—Second step in forming the meatal flap.

part of the time, and is shown in Fig. 92. The auricle is now turned somewhat backward and by continuing the sawing motion a round or oval flap is fashioned out of the concha (Fig. 93). This should be about as large as the ear will permit. If its removal leaves the opening round or oval and the crus of the helix is not invaded,

Fig. 94.—Meatal flap sutured in position.

there will be no deformity. In making these incisions when the sinus or dura is exposed, great care should be exercised. It is a good plan to have the assistant cover them with some flat instrument, or a probe-pointed knife may be used after the incision is started. The ear is now held forward and the flap drawn out behind the auricle.

It will be found to consist of skin, cartilage and fibrous tissue. The dermal layer is carefully dissected from the other parts of the flap, which are then removed, taking care not to cut too deeply into the cartilaginous framework of the auricle. The skin is now sutured to the back of the auricle and tissues adjacent with chromicized catgut (Fig. 94). Care should be taken that the first suture is near the external end of the skin flap and that it draws it well up out of the meatus. Another suture is then placed more internally and the internal tip of the flap is applied to the superior surface of the radical cavity, being of course continuous with whatever integument has been left on the anterior canal wall.

Other Flaps.—The Panse flap is T-shaped, the top of the T being placed vertically in the concha while the long arm splits the canal tube through the center of its posterior wall. The skin is dissected from the fibrous and cartilaginous tissues, which are removed, while the two skin flaps thus formed are sutured, the one upward, the other downward. Korner makes two incisions in the tube, the one above, the other below. By splitting the tube in this way its posterior surface forms a tongue attached at the concha. This is then sutured to the raw surface on the back of the auricle. Besides these there are various modifications of the Panse flap and others which, while ingenious, need not be described, as the one given above in detail will be found to answer all requirements.

After-treatment.—Two modes of procedure from this point will be described, first, without a skin graft, and second, with a primary skin graft. The radical cavity is packed with iodoformized gauze, the end of which is brought out of the meatus. The posterior wound is completely closed with clamps or silkworm-gut sutures and the dressing applied as in the simple mastoid operation. The wound is dressed through the meatus and the rules, during the first two weeks, for changing the gauze packing, removal of the clamps, etc., are practically the same

as in the simple mastoid operation. After this, one must bear in mind that dermatization of the cavity is desired. Granulations may therefore be quite troublesome, as they interfere with this result. They should be kept down with the fused bead of nitrate of silver and the sharp ring curette. Some cavities do better with firm packing, others, if left open. Packing may interfere with the progress of the dermatizing line, although it may keep the granulations down. Large granulations frequently form at the upper posterior angle of the meatus which must be removed with the curette and their bases cauterized with the fused silver bead. A small granulation in an otherwise normal cavity is capable of producing considerable discharge. Granulations may form on the internal tympanic wall and fill the cavity to a greater or less degree, then in this state the surface of the granulating mass may become dermatized. Naturally the fact that this pad of granulations exists over the oval and round windows interferes with hearing. At times this new-formed tissue will break down on account of its lowered vitality and leave a fairly good cavity with better hearing. These patients are discouraging to treat, as one may watch an unsatisfactory cavity developing from day to day and not seem to be able to do anything to prevent it. Still, many of them heal and become dry in from six to ten weeks, or the process of cure may take longer.

Skin Graft.—It is to shorten the period of healing and to insure the formation of a proper cavity that the method of lining these cavities with skin grafts has been devised. This may be done either at the time of the operation (primary graft) or a week or more later (secondary graft). If primary grafts are used, all bleeding-points are tied, the cavity packed with adrenalin gauze and pads placed over all upon which pressure is made while the grafts are being cut. It is absolutely necessary to have a dry cavity to insure success, and it is also safer. Epithelial grafts are cut from the thigh as thin and as large as possible.

This is done with the razor, which is specially designed for this purpose (Fig. 95). These are placed epithelial side upward on glass slides or metal plates and spread out with a needle (Fig. 96). The packing is now taken from the cavity, the graft moved to the end of the plate and caught at two points and held against the anterior wall of the cavity. The plate is now drawn backward, thus sliding from under the graft, which thus lies spread

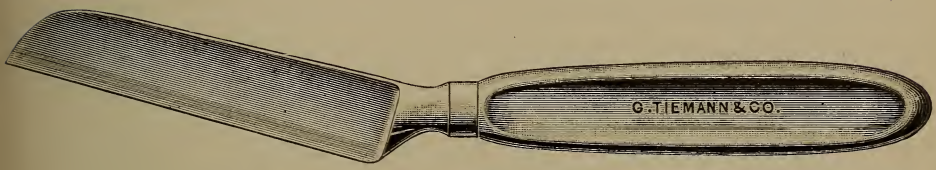

FIG. 95.—Razor for cutting epithelial grafts.

out over the opening of the cavity. It is now tucked in sufficiently to bring its margins in contact with the upper and lower walls of the cavity. A pipette is now inserted between the graft and the inferior canal wall and passed until its tip reaches the internal tympanic wall. The graft is tucked around the pipette so that the cavity beneath the graft becomes air-tight. The assistant now sucks upon the rubber attached to the pipette, drawing out the air and any secretion that has accumulated

FIG. 96.—Teasing needle for Thiersch's grafting.

beneath the graft. This sucks the graft into the cavity. While this is being done the anterior margin of the graft is held in contact with the anterior wall of the canal, that this part of the graft may not slip into the cavity and thus fail to cover the anterior wall which is bare internal to the end of its cutaneous covering. If this maneuver is successful, the graft becomes closely applied to the walls of the cavity. Small pledgets of cotton

dusted with aristol are now applied against the graft; the first, with a black thread attached to facilitate removal, is placed well into the orifice of the Eustachian tube, and the remaining as the cavity may demand to hold the graft closely applied to its walls. Another graft may be applied to the raw posterior surface of the auricle and brought out through the meatus. This will prevent troublesome granulations from forming at the margin of the conchal incision. Only that part of the posterior raw surface of the auricle should be covered that will, when the ear is sutured in position, overlie the radical cavity. If the graft lies between the flap and the surface of the bone posterior to the cavity, it will interfere with healing of the posterior incision. The remaining part of the cavity is packed with sterile gauze, the end of which lies near the meatus and the posterior incision closed with clamps. The remainder of the dressing is the same as after the simple mastoidectomy. The thigh wound, from which the Thiersch grafts have been taken, is covered with silver foil and a dressing applied.

After-treatment.—This operation completely lines the cavity with epithelium, and if the graft takes and there are no contraindications, the gauze and pledgets may remain for four days. The epithelium peels off a day or so later and leaves a dermatizing surface. The bandage should be left off as soon as the pledgets are removed. The clamps are removed from the posterior wound in two or three days; after four or five days, the dressing is held in place by painting with collodion. The cavity is to be irrigated with saline or bichloride, 1 to 10,000, every three to six hours, according to the amount of discharge. The cavity is wiped out daily with peroxide, one-half strength, dried, then alcohol applied, after which boracic acid may be insufflated. If the graft takes in part only, the granulations must be kept down to allow dermatization to proceed. It is a bad plan, however, to pack these cavities, as the grafts may thus be destroyed.

If complications arise during the first four days, it

may become necessary to remove the grafts, or at times the packing and pledgets may be taken out. This must be left to the individual judgment of the surgeon. If he believes that the grafts and the firm packing holding them in place are producing labyrinthitis, he will remove them just as he would redress a wound in which they are not present under the same circumstances. For the complications following the radical operation, the student is referred to Labyrinthitis, Meningitis and Sinus Thrombosis.

Posterior Openings.—At times, after a radical operation, the posterior wound becomes infected and breaks down. In this event it may become necessary to insert a gauze drain into the wound. This may be done temporarily, but it is a bad plan to continue the use of this posterior packing, as to do so results in the formation of a permanent opening brought about by the inward growth of the epithelium from the margins of the wound.

The proper procedure is to pack the cavity through the meatus, place a light dressing over but not into the posterior opening, then by applying a pad anterior to the auricle, the bandage is put on in such a manner as to force the ear upward and backward, thus approximating the margins of the posterior wound. By persisting in this treatment healing will occur without the opening becoming permanent.

Occasionally one is called upon to close these posterior openings for the sake of the cosmetic effect. It then becomes necessary to perform a plastic operation. Several of these have been devised. The object is to form a flap which may be turned into the fistulous opening in such a manner that the epithelial side will become part of the lining of the radical cavity. To accomplish this a tongue of skin may be cut and reflected into the opening, its margins being held in place by sutures (Mosetig-Moorhof), or flaps may be formed on either side of the opening turned inward and held together with sutures (Passow-Trautmann), the raw surfaces being covered

with flaps of integument or allowed to dermatize without them. A modification of these methods may be necessary to suit individual cases and may with safety be left to the ingenuity of the surgeon.

Results.—A properly executed radical operation should result in a dry ear in about 70 per cent. of the patients, most of the other thirty should be nearly dry or only run at intervals, usually from tubal infection. Moreover, the danger to intracranial complications is practically eliminated. The mortality from the radical operation should not be much over 2 per cent., that is, when the operation is done for chronic suppuration alone. If it is a part of the surgical interference for a brain abscess or meningitis, and death results, it can in no way be attributed to the radical operation.

In some patients the hearing is made worse by the operation, in others the hearing is not changed, while occasionally one sees a patient whose hearing is improved. As a rule the more acute the hearing before operation, the greater the danger that the patient will be deafer after it. Marked impairment of hearing is not so often changed for the worse. The uncertainty of prognosis as to the hearing makes it a rule in binaural suppuration that the worst ear should be operated on first, and the second ear should not be operated upon if it can possibly be avoided, unless the hearing in the operated ear becomes better than that in the other ear.

MODIFIED RADICAL OPERATION.

Indications.—Several modifications of the radical operation are performed and advocated by different surgeons. The principal reason for them is the desire to preserve the hearing. It is claimed that by leaving the drum membrane and ossicles, the hearing will be much better than if these structures are removed. This may be conceded if their relations are not disturbed during the operation or their movements limited by subsequent

formation of cicatricial tissue during the healing process. One is never certain that all of the diseased tissue has been removed, as so much of the cavity is not open to inspection. Moreover, for the operation to be at all better than the radical procedure, in regard to the conservation of hearing, the drum membrane and ossicular chain must be of some value in the conduction of sound. Many radical operations are done when this is not the case, these structures rather interfering with, than aiding in, sound conduction. While all diseased tissues may not be removed, the cavity, as usually formed, is so small that during the healing process it fills with granulations, troublesome to deal with and usually interfering with ultimate success. This modified procedure is indicated when one ear is very deaf and it becomes necessary to operate upon the opposite ear which possesses a fair amount of hearing.

Operation.—The posterior incision and exposure of the canal are practically the same as in the radical operation. The lining of the osseous canal usually tears near the membrana tympani so that the part of the canal adjacent to the tympanic ring which it is proposed to leave is not denuded of epithelium. The antrum is opened the same as in the radical operation, and its walls curetted and all granulations removed. The superior wall of the canal may be taken down except in proximity to Shrapnell's membrane, where a substantial rim is left. The vault is opened by removing the cells, if present, between the cortex of the superior canal wall and the inner table. The vault is now cleared of granulations and diseased membrane as far as possible without injuring or displacing the ossicles. The posterior canal wall is taken down except in the region adjacent to the tympanic ring. In fact, throughout the entire operation care is taken not to injure the drum membrane or ossicles. The meatal flap is cut and sutured in position as in the radical operation. Skin grafts are not as a rule inserted. The auricle is sutured in position and the external end of the gauze

which has been packed into the operation cavity is brought out through the meatus. This is changed in three or four days, and reinserted daily for a few dressings. Granulations are dealt with as in the radical operation without skin graft. If the operation is successful, the bone wound becomes dermatized and free from secretion. If this does not occur, the radical operation may be subsequently performed.

OTITIS MEDIA PURULENTA RESIDUA (O. M. P. RESID.).

Definition.—After a middle-ear suppuration has exhausted itself or been cured by non-operative treatment, certain changes are left or residual. These changes may be slight and interfere very little with the function of the organ of hearing or may be more marked and result in deafness or tinnitus. While these residual conditions are usually found as a result of chronic suppurations, they may also occur after the acute form. It seems best, therefore, to call them Otitis Media Purulenta Residua (O. M. P. Resid.), leaving out the word chronica which has usually been inserted.

Causation.—The condition is caused by suppuration of the middle ear, and whether or not changes will be left which will produce symptoms depends largely upon the intensity of the original process and the location and kind of these changes themselves.

Pathology.—There is regularly more or less destruction of the membrana tympani, which has usually been repaired to the best of nature's ability by cicatricial tissue. If a small defect, this may be quite complete, the membrane regaining its motility and usefulness. In large perforations the membrane in healing usually becomes more or less adherent to the internal tympanic wall. Cicatricial bands and adhesions may be present that interfere with the motility of the ossicular chain, or the ossicles may have been destroyed by disease, in whole

or in part. In some instances the perforation fails to heal, but the ear continues dry.

Symptoms.—The patients come complaining of deafness or of tinnitus or both. The deafness may be of a mild degree, the patient seeking relief for the head noises. Not infrequently the patient states that when his ears were discharging he heard better than since they were dry. This is usually found in patients with posterior defects in the drum membrane, and is explained by the fact that as long as the mucous membrane is moist it is pliable, but when it becomes dry and the fibrous tissue contracts, it becomes more rigid. This condition around the capitellum of the stapes, the ossicular chain or in the niche of the round window, would account for this symptom. The functional tests show the impairment of hearing to be due to lesion of the sound-conducting mechanism unless the labyrinth is involved also. This occasionally occurs but cannot be considered as part of the process under discussion, although in a sense it may be residual, usually being a result of a labyrinthine inflammation. Changes in the labyrinthine capsule or inner ear described under Otosclerosis may occur in a residual ear and the tests would be modified accordingly. Less often a full feeling is a symptom. A patient recently observed complained of the snapping of the cicatricial membrane which had covered in the Eustachian tube, and he was continually swallowing to overcome the annoyance, being very anxious concerning the entotic noise.

Otoscopic Appearance.—The changes in the membrana tympani are those which have been brought about by the exhausted suppuration. A perforation may exist which has failed to heal. The size and location of these perforations vary considerably, although they are usually not large. Their failure to cicatrize is brought about by the epithelium growing over the fibrous layer and meeting the mucous membrane, thus preventing the margins of the perforation from throwing out new tissue to close or contract the opening. Usually, however, the

defect is closed by cicatricial tissue which is adherent to the internal tympanic wall, especially if the original perforation was a large one. Where this tissue is formed on the inner wall of the middle ear, it is frequently covered with epithelium which presents a well-marked luster, not very unlike that of the membrana tympani. In large posterior defects that have thus healed, it is often possible to make out the landmarks on the inner tympanic wall, namely, the niche of the round window, the promontory, the oval window with the stapes in position, perhaps also the long process of the incus. The drum membrane anterior to these defects may be either cicatricial or the original membrane. The manubrium is very frequently adherent to the inner tympanic wall and may or may not be displaced or rotated.

Diagnosis.—The diagnosis is made by the occurrence of impaired hearing or tinnitus, with the changes in the drum membrane which show an exhausted suppuration. Small perforations which have healed with cicatricial tissue may very closely resemble the depressed areas seen in O. M. C. C. The history will usually enable one to decide, but occasionally one sees drum membrane changes which could only have been produced by a former suppurative process and be unable to obtain any history of such a process, it having occurred in infancy or childhood and therefore not having come to the patient's knowledge. When there is a perforation which has not closed and the ear remains dry, it may be considered as O. M. P. Resid. If, however, at intervals such a process is lighted up and discharges, the word "residual" seems out of place. To call it a recurrent O. M. P. C. or an otitis media in a residual ear seems more reasonable.

Prognosis.—The occurrence of O. M. P. Resid. may be regarded, in one respect, as fortunate, as it represents the healing of an active process. Nevertheless, the outlook as to much improvement of the hearing or relief from the noises cannot be regarded as very favorable.

Treatment.—The treatment of a resolving purulent process of the middle ear with a view to minimizing

impairment of hearing, has already been discussed. If the perforation has been small and become healed, inflations sometimes have a favorable influence on the deafness. With the view to improving the hearing, attempts have been made to bring about the healing of perforations which remain open. Probably the best method is by applying a paper splint by moistening a small piece of waxed paper and placing it over the opening in the membrane. In many instances this produces marked improvement of hearing at once. The parts should be inspected every few days to see that the paper is in position and that reactionary signs have not developed. If there is inflammation or discharge, the paper should be syringed from the ear, and this allowed to subside before reapplying the splint.

In large perforations pneumomassage may be tried with prospect of slight improvement in the occasional patient. It is sometimes possible to increase the hearing by the insertion of a pledget of cotton. The absorbent cotton is wound upon an applicator in such a manner as to produce a fluffy end with a long stem, which is the part wrapped around the applicator. The pledget is slipped from the instrument and the fluffed end molded to the proper size. A few drops of liquid petrolatum are allowed to soak into the cotton, which is then inserted into the meatus by means of the stem. It is to be placed in contact with the inner tympanic wall in the region of the stapes and is intended to press upon this ossicle and conduct sonorous vibrations in an amplified form. Patients soon learn to introduce and remove these pledgets of cotton and become very skilful in placing them in the exact position to improve the hearing, which it does at times to a remarkable degree. One would naturally suppose that the division of adhesions and bands which produce rigidity of the ossicular chain or interfere with the motility of the stapes would result in improvement of hearing, but operations in these patients have been disappointing, although occasionally of benefit.

CHAPTER X.

SUPPURATIVE DISEASES OF THE LABYRINTH.

Under the heading of Suppurative Diseases of the Labyrinth are discussed those diseases of the labyrinth which are usually seen as complications of middle-ear processes of a suppurative nature.

FUNCTIONAL TESTS OF THE STATIC LABYRINTH.

Functional tests have already been described by means of which the condition of the acoustic labyrinth and its central connections may be determined. It now remains to describe certain tests, whereby one may derive information as to the condition of the static labyrinth. In doing this an effort will be made to divest the subject of much of its intricacy, and it is therefore necessary to avoid problems still unsettled as well as those which, while interesting in themselves, would tend to confuse the student.

Physiology.—For all practical purposes, the cristæ ampullæ are the end-organs of the vestibular nerve. While filaments of this nerve also end in the cristæ of the vestibule, very little is known of their function. The cristæ ampullæ are little mounds of nerve tissue in the dilated ends of the three membranous semicircular canals, surmounted with hair-like processes which, projecting into the lumen of the canal, are influenced by movements of the endolymph contained in the membranous labyrinth. The nerve fibers from these cristæ are connected with the vestibular nuclei in the medulla and mainly through them with the cerebellum, a spinal nucleus and with the motor oculi nucleus situated in the floor of the aque-

duct of Silvius. Impulses arising in the cristæ produce movement of the eyes through the latter connection. The motor oculi nucleus enervates the eye muscles, and the connection is so formed that muscles which move the eyes in a given direction receive their enervation from the same part of the nucleus. For example: the internal rectus of one eye and the external rectus of the other eye are enervated from the same part of the same nucleus. (Fig. 97). To move the eyes to the left the internal rectus of the right eye and the external rectus of the left eye contract and the stimulus comes from the right motor oculi nucleus. Now, if the right nucleus is stimulated by

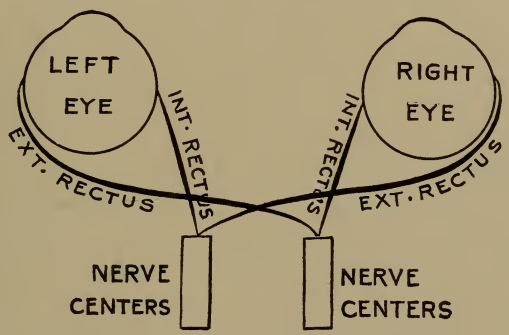

FIG. 97.—Diagram illustrating the origin of the eye muscles.

impulses received through the connecting fibers between it and the cristæ ampullæ it produces movement of the eyes to the left. This movement takes place without volition, being simple a result of the stimulation of the cristæ. As soon as the subject becomes conscious of this involuntary movement of the eyes to the left, which he does by the apparent movement of objects to the right, by an effort of the higher centers he brings the eyes back to the normal position. The movement toward the left produced by the vestibular irritation is slow, the movement to the right caused by impulses emanating from the higher centers is a rapid one. A succession of

these to-and-fro movements is called nystagmus, and as it is due to stimulation received from the vestibular nucleus it is called vestibular nystagmus. It differs from ocular nystagmus, that is, nystagmus due to disease of the eyes, as well as almost all other forms of nystagmus, in that in vestibular nystagmus there is a slow movement in one direction and a rapid movement in the opposite direction, that is, a slow component and a rapid component. It may be produced by disease of the static labyrinth or the cerebellum, although the latter is not as typical as the former. When caused by disease it is called spontaneous nystagmus. It may also be produced by certain experiments to which the patient may be subjected and is then known as experimental or induced nystagmus. In the normal patient it may be experimentally induced in three ways, namely: (1) by rotating the patient; (2) by syringing the ear with hot or cold water; (3) by the galvanic current.

Ewald's Laws.—In order to understand either rotation or caloric nystagmus and be able to predict the form and direction of the eye movements which will follow any rotation or syringing, Ewald's experiments and the rules derived from them must be thoroughly understood. Ewald drilled a small hole in the semicircular canal of pigeons, and attached a delicate tube connecting with a rubber bulb (Fig. 98). The end of the canal away from the ampulla he plugged with lead. It will readily be seen that pressure upon the bulb would produce a current in the canal toward the ampulla, while relaxation of the bulb would produce a current away from the ampulla, that is, canalward. When this apparatus was attached to the horizontal semicircular canal and the bulb compressed and a current toward the ampulla produced, the pigeon turned its head and eyes to the opposite side. When the pressure was relaxed and the current reversed, that is, canalward from the ampulla, the head and eyes moved to the same side but less strongly. To state this in the form of a law: (1) *In the horizontal semicircular canal*

when the current is from the canal toward the ampulla, the crista becomes plus (+). In like manner the superior semicircular canal was experimented upon but, unlike the horizontal, the greater movement occurred when the current was away from the ampulla, that is, canalward. (2) *In the superior semicircular canal when the current is from the ampulla toward the canal, the crista becomes plus.* The posterior is the same as the superior.

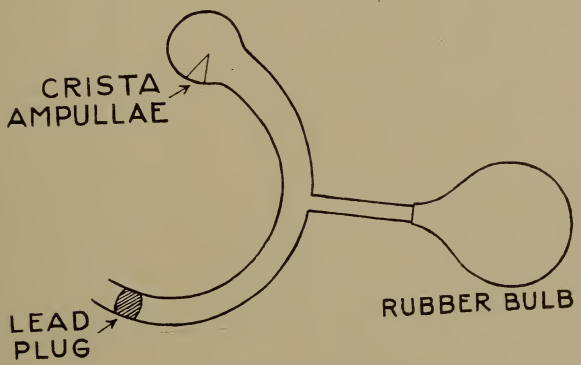

FIG. 98.—Ewald's experiment.

Rotation Nystagmus.—By rotation, currents in the semicircular canals may be produced in man, and by observing the ensuing reactions a conception of the condition of the static labyrinth may be formed. The patient is placed in a chair capable of rotation. When he is seated with head erect, it is evident that the horizontal canals lie, approximately, in the plane of rotation. He is now rotated ten times. At the beginning of rotation the endolymph in the horizontal semicircular canals, by reason of its inertia, lags behind. This has the effect of producing a current opposite to the direction of rotation. This produces a nystagmus which follows the rules to be mentioned, and has been seen by an observer occupying a platform which rotated with the patient, thus allowing a view of the eyes to be obtained during the rotation. But

this method is impracticable, therefore not used. After the patient has been rotated ten times, the endolymph has picked up the motion of the horizontal canals and is moving at the same rate and in the same direction as the canals and there is therefore no current. The rotation is now suddenly stopped and the canal becomes at rest, but the endolymph on account of its inertia continues in motion. This produces a current in the direction of rotation. As the ampulla of the horizontal canals lies anteriorly it follows that the current will be toward the ampulla in one and away from the ampulla in the other (Fig. 99). While the current in the two canals, the one plus and the other minus, have the same effect upon the eyes, since the

Fig. 99.—Diagram illustrating an inertia current in the horizontal semicircular canals.

impulse from the minus current is added to and augments the reactions arising from the plus current on the opposite side, it is best in figuring the reactions to think only of the crista which is plus. Thus in turning from left to right, as in Fig. 99, the movement of the fluid after rotation in the left canal is toward the ampulla, and as the head is erect this must be the horizontal canal and it is therefore plus according to rule (1). By completing the diagram (Fig. 100), it will be seen that a stimulus goes to the central nuclei of the vestibular nerve from the crista of the left semicircular canal; thence to the motor oculi nucleus on the left side. In this nucleus an impulse is aroused which produces contraction of the internal rectus on the left

side and the external rectus on the right side. This turns the eyes to the right. But the higher centers send out an impulse to the opposing muscles which, with a quick movement, bring the eyes back to their normal position, that is, the quick component is to the left. This sequence of events is repeated again and again during from twenty to

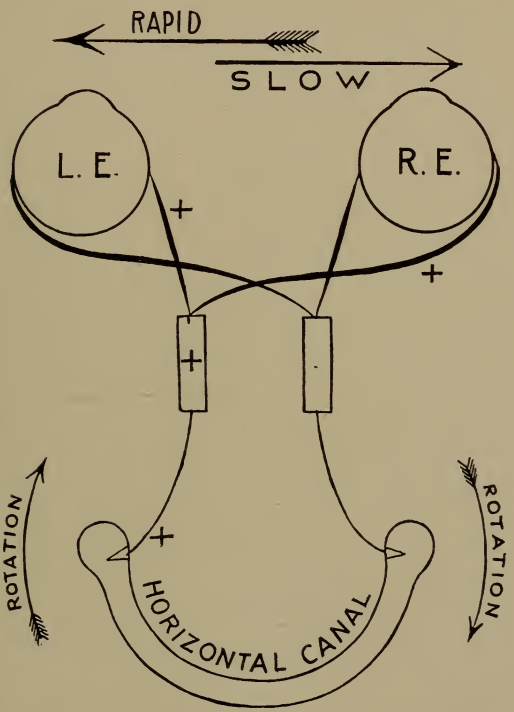

FIG. 100.—Diagram for rotation to right with head erect = horizontal after nystagmus to the left.

thirty seconds in the normal labyrinth. The result of the experiment is horizontal nystagmus to the left after rotating the patient to the right. Without entering into a discussion as to the exact origin of the impulse producing the quick component, it may be stated that it does not take place under anesthesia or when for any reason

the patient is unconscious. Nystagmus is arbitrarily named from its quick component. Thus the nystagmus in the above experiment is called to the left as the quick component is to the left, the slow being to the right. A nystagmus is always intensified by having the patient look in the direction of its quick component. Therefore,

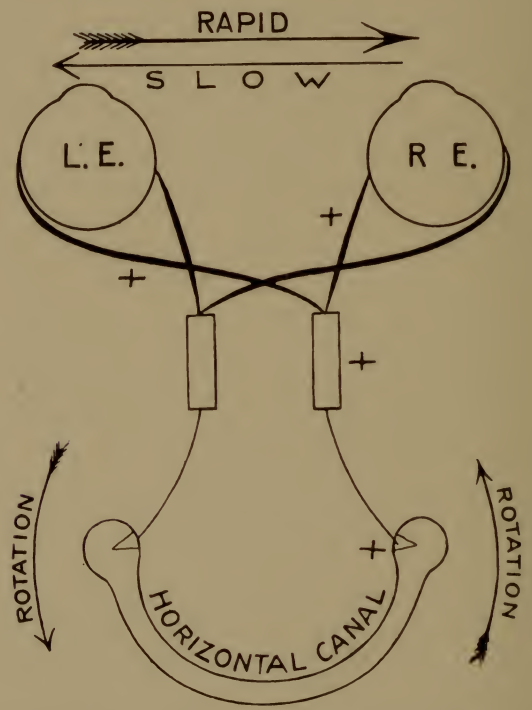

FIG. 101.—Diagram for rotation to left with head erect = horizontal after nystagmus to right.

in eliciting this nystagmus, the patient would be directed to look at the finger held as far to the left as possible without causing the bridge of the nose to interfere with the view of the right eye. In determining the direction in which one is rotating a patient, one must think of the patient's face. For instance, if the patient's left eye is

following his right eye, he is being rotated to the right and *vice versa*.

Suppose the patient be rotated to the left with head erect. The diagram is shown in Fig. 101. The right eye follows the left eye during rotation. At the end of ten revolutions and after the head is at rest, the endolymph current is in the direction of rotation, as shown by barbed arrow. That is, toward the ampulla in right horizontal canal which thus becomes +; right centers +; internal rectus of right eye and external rectus of left eye +. Slow movement of eyes, shown by plain arrow to left; quick movement shown by barbed arrow to the right = horizontal after nystagmus to the right produced by rotating patient with head erect to the left.

Suppose the head be bent forward 90 degrees and the patient rotated to the right. This brings the superior canals into the plane of rotation. This statement is not strictly correct, as the superior semicircular canals are at an angle of about 45 degrees with the coronal plane. Still, as both of their ampullated extremities are situated externally, the current of endolymph which is produced by rotation is practically the same as though they were in the coronal plane. The nystagmus produced by rotation with the head forward 90 degrees is rotatory. The law determining the form of nystagmus is that the eye movements will be in that meridian in which the horizontal plane cuts the cornea during rotation. Thus, with the head erect the horizontal plane cuts the cornea perpendicular to the median line and rotation produces horizontal nystagmus; with the head bent forward or backward 90 degrees, the horizontal plane cuts the periphery of the cornea and rotation produces rotatory nystagmus; while with the head 90 degrees to one side the horizontal plane cuts the cornea in the sagittal plane and rotation produces vertical nystagmus. The law also holds true in intermediate positions of the head.

It is evident that the eye muscles which produce rotatory nystagmus are not the same as those which

produce the horizontal form; nevertheless, the diagram, although perhaps slightly inexact, will work out to a correct conclusion without involving one in more difficulties. Rotatory nystagmus is named from the direction of the quick component of the upper end of the vertical meridian of the cornea. See Fig. 102, which shows rotatory nystagmus to the right, the upper pole of the vertical meridian having a quick movement toward the patient's right, the slow component being to the patient's left. With the head forward the ampulla of the superior semicircular canals become posterior as well as external. Drawing the diagram (Fig. 103), if the patient is rotated to the right, current at the end of rotation to the right,

Fig. 102.—Diagram illustrating the determination of the direction of rotatory nystagmus.

that is, toward the ampulla in the right canal and away from the ampulla in the left. Left crista +, according to Ewald's law (No. 2), left centers +, muscles which rotate the upper pole of the cornea to the right +, therefore, slow component to the right; rapid component to the left =. Rotatory nystagmus to the left produced by rotating the patient with the head forward 90 degrees to the right.

Suppose the patient with head backward 90 degrees is rotated to the right. The ampulla of the superior semicircular canals which are below and external with the head erect are thus brought to an anterior and external position and the cristæ are therefore stimulated by the

current produced by rotation, which is toward the ampulla in the left canal (Fig. 104). Right crista +, right centers +, slow component to the left, rapid component to the right =. Rotatory nystagmus to the right produced by rotating the patient to the right with head backward

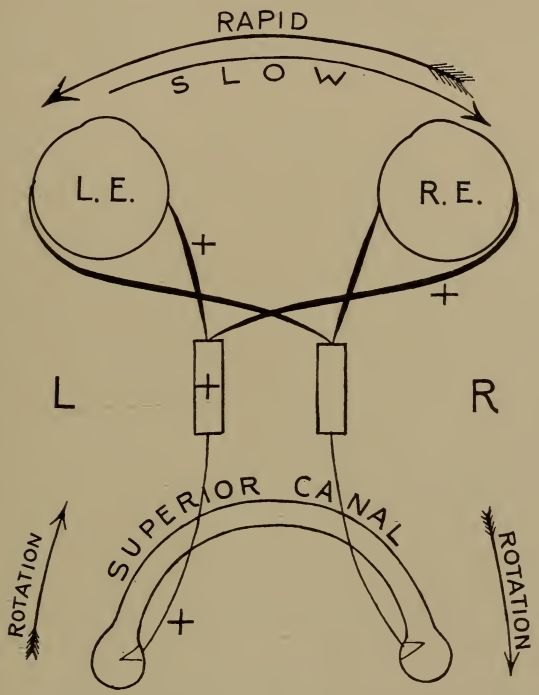

FIG. 103.—Diagram for rotation to right with head forward 90° = rotatary after nystagmus to left.

90 degrees. It is only necessary to remember Ewald's laws which are founded on physiological facts. They are true but no reason can be given why nature has designed these canals in exactly this way. By drawing his diagram, the student should now be able to figure out the effect of any given rotation.

Caloric Test.—In the rotation experiments the current is produced by the inertia of the endolymph. In the caloric tests a gravity current is produced by means of heat or cold. With the head erect the superior semicircular canal is above the vestibule (Fig. 105). If, therefore,

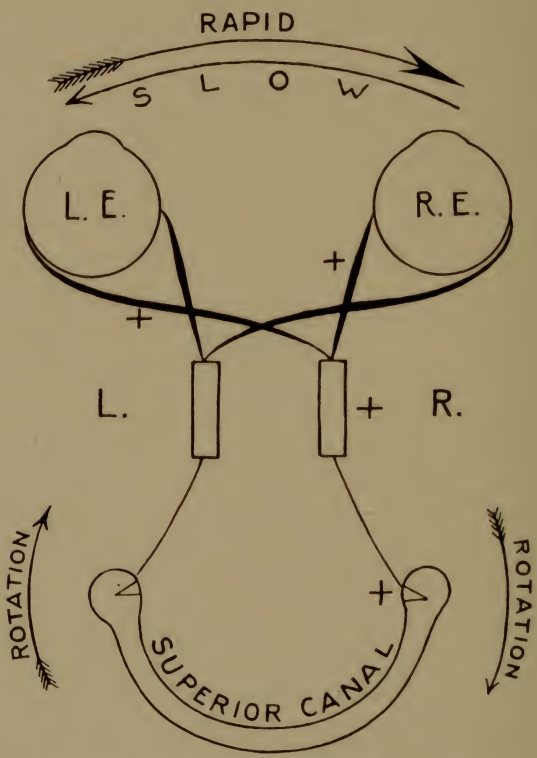

FIG. 104.—Diagram for rotation to right with head backward 90° = rotatory nystagmus to right.

a stream of cold water, say at a temperature of 50° F., is directed against the membrana tympani, the air in the tympanic cavity becomes colder and this change of temperature is communicated to the vestibular fluid adjacent to the internal tympanic wall. It thus becomes condensed, and gravitating, produces a current downward.

This draws fluid from the superior semicircular canal, producing a current toward the ampulla as shown in Fig. 105. According to Ewald's law, this makes the canal minus. The opposite crista would therefore be plus, as normally the stimuli emanating from the opposite labyrinths are exactly balanced and this syringing with cold water creates an imbalance or disharmony.

Suppose the right ear be syringed with cold water, with head erect (Fig. 106). This produces a current

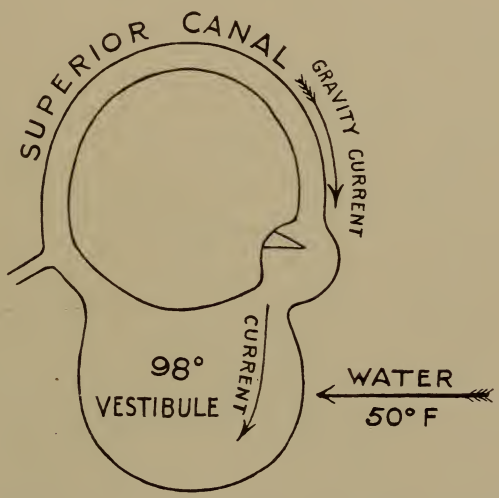

Fig. 105.—The method of the production of the gravity current.

toward the ampulla in the right superior canal. Right canal —, left canal +, left centers +. Slow component to the right, rapid component to the left = rotatory nystagmus to the left produced by syringing the right ear with cold water with the head erect.

Suppose the right ear be syringed with hot water. This usually produces less effect than cold, as it is impossible to use water as many degrees above the normal temperature of the labyrinthine fluid as can be used below with cold water. Thus, water at a temperature

of 50° F. will produce more effect, other conditions being the same, than water at 115° F., the difference being as 49 to 16, which is the amount these temperatures vary from the temperature of the labyrinthine fluid, say 99° F. As the hot water flows into the meatus it raises the temperature of the labyrinthine fluid adjacent to the

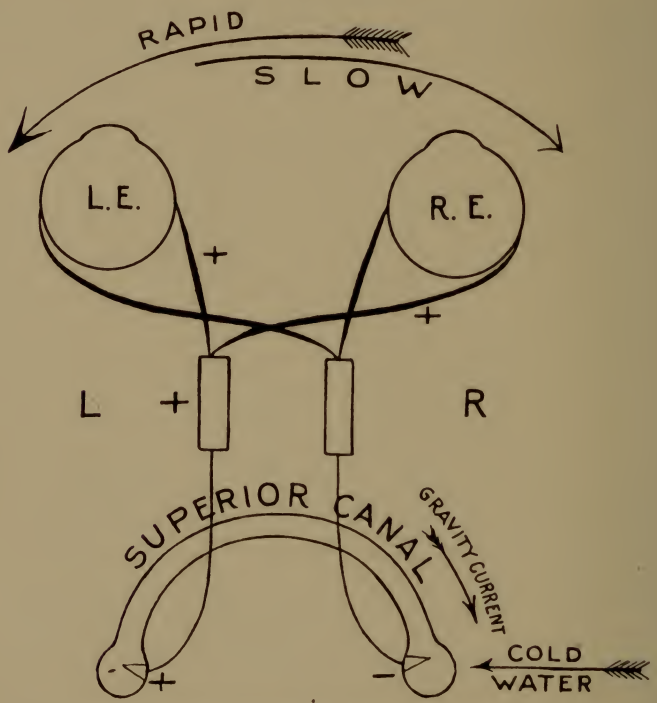

Fig. 106.—Diagram for syringing the right ear with cold water, head erect = rotatory nystagmus to left.

internal tympanic wall, and it being of a less specific gravity, rises into the superior semicircular canal, producing a current away from the ampulla, that is, toward the canal (Fig. 107). Right crista +, right centers +, slow component to the left, rapid component to the right = rotatory nystagmus to the right produced by syringing

the right ear with hot water. Let the student draw his diagrams and work out the result of syringing the left ear with hot or cold water and when the body is inverted with head downward.

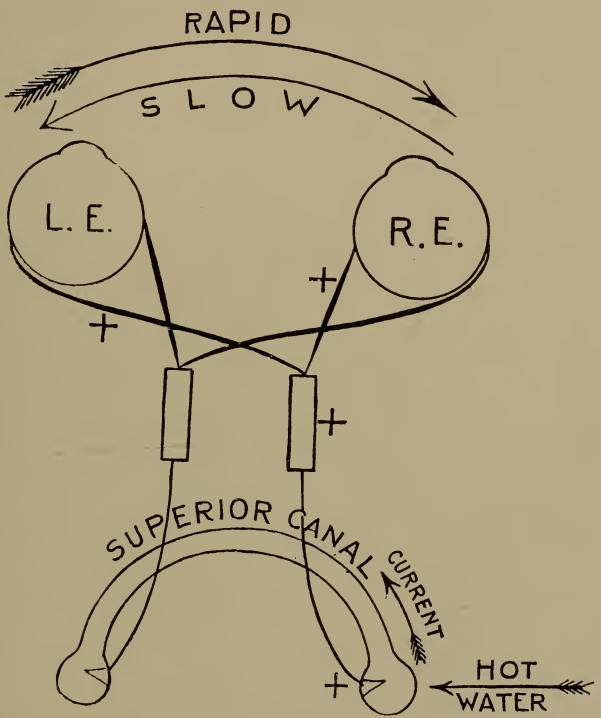

FIG. 107.—Diagram for syringing the right ear with hot water with head erect = rotatory nystagmus to right.

If the patient lies in the recumbent posture with the head backward at an angle of 60 degrees with the vertical plane, in which position the horizontal semicircular canal is most favorably situated to be stimulated by a gravity current, as it then becomes vertical, its ampulla being at its upper extremity, syringing with cold water produces a current away from the ampulla and the canal

becomes minus. Suppose the right ear be syringed with cold water (Fig. 108). Current away from the ampulla in right canal, right crista —, left crista +, left centers +, slow component to the right, rapid component to the left = horizontal nystagmus to the left produced by syringing the right ear with cold water with the head

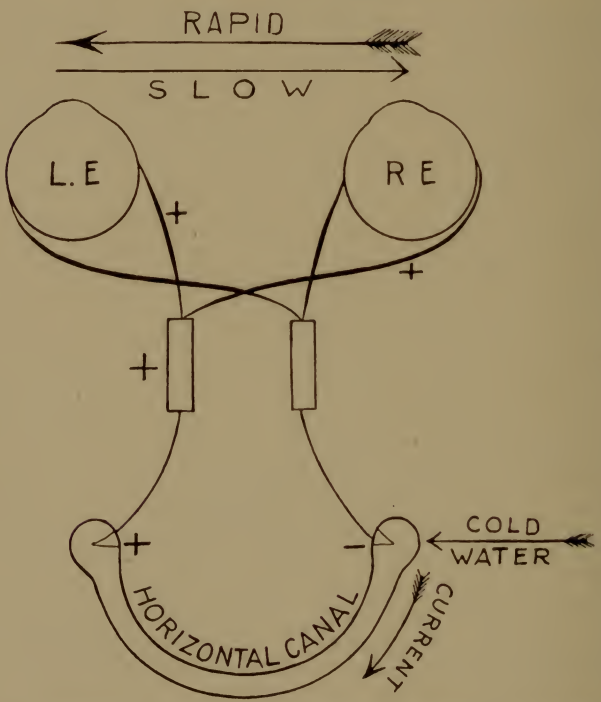

FIG. 108.—Diagram for syringing the right ear with cold water, head backward 60 degrees = horizontal nystagmus to left.

backward at an angle of 60 degrees. Let the student figure the reactions which would occur if, in this position, the right ear is syringed with hot water or the left ear syringed with hot or cold water.

When the ear has been syringed with hot or cold water and nystagmus produced, if, during the continuance

of the eye movements, the position of head is changed, the nystagmus is also changed. For instance, with the head in the optimum position for stimulation of the horizontal semicircular canal, that is, with the head backward at an angle of 60 degrees with the vertical plane, the right ear is syringed with cold water and there results a horizontal nystagmus to the left as in Fig. 108. If now the head is changed to the vertical position and tipped slightly to the right, that is, the optimum position for stimulation of the right superior canal, the gravity current will affect the crista in the superior semicircular canal, the current being toward the crista, from the canal, as in Fig. 106, and there is produced a rotatory nystagmus to the left. The nystagmus has been changed from horizontal to rotatory by changing the position of the head. This has been caused by change in the crista stimulated and is an argument in favor of the theory that the nystagmus persists only during the continuance of the endolymph current.

Galvanic Test.—The induction of nystagmus by the galvanic current offers no special advantage over the rotation or caloric tests, nor is it as generally employed. Galvanism produces a combined rotatory and horizontal nystagmus which shows that all of the cristæ, perhaps the nerve as well, are acted upon. No amount of pains has enabled the stimulation to be limited to one canal, to the exclusion of others. In patients from whom the labyrinth has been removed, if the vestibular nerve has not degenerated, nystagmus may still be produced. The nystagmus is in the direction of the current. For instance, if the cathode is applied at the right, and the anode at the left ear, the nystagmus will be to the right as the current is from the anode to the cathode. In this experiment a current of about 4 ma. will produce nystagmus. It will be noticed that in passing through the head both labyrinths and nerves are acted upon, and made in this way the galvanic test resembles the rotation test which also acts upon both labyrinths.

The anode may be held in the hand or placed elsewhere upon the body and the cathode placed before or back of the auricle. This will produce nystagmus toward the side to which the cathode is applied. A stronger current will be required. This acts upon one labyrinth alone, and in this respect resembles the caloric test, although unlike this test the canals cannot be separated. The degree of irritability is shown by the amount of current required to produce nystagmus. If the cathode is divided and one-half placed at either ear, no nystagmus is produced if both labyrinths are alike in their irritability, the stimulation of one being equal to and opposite in effect to the stimulation of the other.

Fistula Test.—The fistula test should be active only in an ear in which there is a defect in the labyrinthine capsule, although occasionally there is some response to this test in an ear not thus diseased. The test is made by inserting the tip of a Politzer air-bag into the meatus and compressing and relaxing it, or if there is a mastoid wound, a Bier's cup to cover all. This increase of pressure would act upon a fistula by compressing the membranous labyrinth and producing a current in the endolymph. More often than in any other situation, there is a fistula in the horizontal semicircular canal. Compressing and rarefying the air would act very much as in Ewald's experiments. On compressing the bulb the force would act upon the membranous canal, the lumen of which would thus become narrowed and an endolymph current toward the ampulla produced. On relaxing the pressure the current would be in the opposite direction. The nystagmus produced by the fistula test is not continuous. The regular reaction is that on compressing the bulb the eyes are directed to the opposite side and brought back with a quick motion. On relaxing the bulb the slow movement less in degree is to the side experimented upon, while the quick movement is to the opposite side. This is considered the regular reaction, and if the test is positive, is more often present than any

other; but there are various other reactions present at times. If upon compressing or relaxing the bulb there are any involuntary movements of the eyes, slow in nature, which are corrected by opposing quick movements, it is safe to say that the fistula test is positive.

Value of Tests.—The object of the various tests is to determine whether or not a given labyrinth is active, and if possible the degree of its activity. In performing the rotation test with head erect and taking the time from the instant the rotation is stopped until the nystagmus ceases, the usual time in the normal person is between 20 and 30 seconds, depending somewhat upon the rapidity of the rotation. If, after the two rotations in opposite directions, the nystagmus is about the same and between 20 and 30 seconds' duration, it is assumed that both labyrinths are equal and functionating. If instead of being equal the duration of the nystagmus in one should be, for instance, 20 seconds, while in the other it was less than 10 seconds, it would be reasonable to suspect that one was not functionating; then this patient should be tested with his head forward or backward so that the inertia current would act upon the superior semicircular canals, and if a marked disparity, say of over one-half, in the two sides was shown, one's suspicion would be increased that one side was not functionating. The rotation test for that labyrinth would be negative. If, upon rotation, the duration of the after-nystagmus in either direction was about equal but much less than normal, for instance, between eight and twelve seconds, one would suspect that one labyrinth was dead and that compensation had occurred. Whether or not this was the case might be determined by the caloric test.

Compensation.—When there exists an endolymph current toward the ampulla in the horizontal semicircular canal, for example, an impulse is produced which, transmitted to the central ganglia, causes certain results, among others nystagmus, and the crista is considered to be plus or in the positive phase. If the current is reversed,

that is, canalward from the ampulla, the effect on the eyes is the same but opposite in direction and about one-half as strong. The crista would then be minus or in the negative phase. The negative phase of one crista is in effect the same as the positive phase of the opposite crista and about one-half as much. This is true of all the cristæ and therefore of the static labyrinth as a whole. Now, if one labyrinth is destroyed by disease or operation in such a manner that the cristæ are also destroyed, the negative phase of the active labyrinth increases or its positive phase diminishes in force until a balance is produced, the one labyrinth thus acting in a fairly efficient manner for both. In rotating a patient who has compensation but one labyrinth is influenced by the currents produced, the other being in such a condition that currents cannot be produced, but the reactions are the same in kind, as though both labyrinths were active but differ in degree. One would hesitate to consider compensation present if the duration of the after-nystagmus, with head erect, was over fifteen seconds. After the destruction of one labyrinth by disease or operation some patients develop compensation; others do not in the sense in which the term is here used. It seems that some condition is necessary in the destroyed labyrinth in order that it should take place. Ruttin believes that this lies in the cristæ. If they are destroyed by operation or by the formation of bone or fibrous tissue in a latent suppurative process, then compensation will take place. Until further light is thrown upon the subject, the author is prepared to accept this as true.

Caloric Test.—The caloric test has the advantage of being used upon one ear alone. It settles positively whether the labyrinth under examination can react to gravity currents produced by local change in temperature of the labyrinthine fluid. About the only condition in which doubt may be felt is in patients who already have spontaneous nystagmus. For instance, a patient has a spontaneous nystagmus to the left and it is desired to

ascertain whether or not the right ear is capable of responding to caloric stimulation. The right ear may be syringed with cold water and it may be noted whether or not the nystagmus is increased, or the ear may be syringed with hot water to ascertain whether the nystagmus to the left is diminished or one produced to the right. One may still be in doubt after trying both of these plans. As an aid in such a case a fixator has been devised. This is

FIG. 109.—Barany fixator (Kerrison) with author's permission.

merely a head band to which a rod is attached in such a manner that it may be moved as desired (Fig. 109). At the end of the rod is an object upon which the patient fixes the eyes. The rod is moved until such a position is obtained that there is no nystagmus. The ear is then syringed, and if nystagmus is produced, the caloric test is positive.

Attempts to form a conception of the degree of irritability of the labyrinth by the caloric test are disap-

pointing. It is necessary to determine how much water at a given temperature, flowing through what length of time, is necessary to produce nystagmus in the normal labyrinth. When this is determined a variation is not necessarily due to a difference in the irritability of the labyrinth as many conditions, outside of the labyrinth, may exist to cause the reaction to be early or late in appearing. Thus the nystagmus comes on quickly with exposed inner tympanic wall covered with thin mucous membrane or skin graft. On the other hand, if it is covered with granulations, polypi or a thick bed of cicatricial tissue, the nystagmus is slow in appearing. The same result is produced by stenosis of the external auditory canal. So, while one, after noting the conditions and the time of the appearance of the nystagmus, may reasonably infer in certain instances that the labyrinth is sluggish or abnormally active in its response to caloric stimulation, the attempt to accurately determine the degree of increase or decrease is not apt to be of much clinical value. Those desiring to pursue the subject further are referred to Brünnings[1] and Kaproff.[2]

CIRCUMSCRIBED LABYRINTHITIS.

Causation.—It is usually produced by chronic suppurative processes, especially cholesteatoma, although it may occur in an early stage of a tubercular otitis media.

Pathology.—Circumscribed labyrinthitis, as at present conceived, is a localized sequestration or erosion of the labyrinthine capsule which results in the formation of an osseous defect. The horizontal semicircular canal is the one usually involved, as it seems by its exposed situation to be favorably located to be affected by middle-ear processes, although the other canals are at times involved. The semicircular canals seem peculiarly fitted by their

[1] Verhand. der Deutsch. Otol. Gessel., 1910, and Zeitsch. für Ohrenheilk., 1911.

[2] Passow's Beitrag., vol. ii, p. 190.

anatomical construction to be the seat of circumscribed inflammation. When a bony defect is found in other parts of the labyrinthine capsule, for instance, the promontory, it is the rule to find the labyrinth generally involved, although there are at times exceptions. The process may be limited to the bony capsule or there may be a localized membranous labyrinthitis.

Symptoms.—The disease may have presented symptoms or be found at the radical operation not having been previously suspected. This is more probable to occur if the surgeon is not in the habit of having fistula tests made on all patients before operation. When symptoms are present, vertigo is usually one of them. It is usually of mild type but may be more severe and occur in attacks during which there is nausea and vomiting. It may be increased or brought on by sudden changes in position of the head, as in stooping or turning around suddenly. Exceptionally, a severe form of vertigo may occur, but in this event one suspects that a serous labyrinthitis may be present. There is usually nystagmus directed either toward the involved side or to both sides. It may, however, be absent. It is not as a rule marked, usually being seen in lateral positions of the eyes. It is usually a combination of the horizontal and rotatory types. The hearing is not as a rule affected, any deafness which may be present showing tests of involvement of the conducting mechanism. Exceptionally, when the attacks of vertigo are present, the hearing is diminished from involvement of the perceptive mechanism. One may speculate on the cause of this, but very little is known.

Tests.—Upon applying the tests if the patient has this form of labyrinthitis and no other, there will be found: hearing present, rotation positive, caloric positive, fistula test positive. Many place very little reliance upon the fistula test, claiming that some response is made to it in the normal condition owing to the disturbance of the structures in the oval and round windows caused by the change in air pressure, the test thus being positive without

a fistula, while the conditions may be such in the region of a defect in the labyrinthine capsule that the change in pressure, used in making the test, would not be communicated to the labyrinthine fluid. All of this may be to a certain extent true; nevertheless, the fact remains that as a rule it is positive in fistulæ and should it occasionally appear to be positive in the absence of a fistula, the test is still of value as showing that the labyrinth possesses irritability.

Diagnosis.—Should a patient with a suppurative process in the middle ear have vertigo mild in degree, nystagmus to the same or to both sides in lateral positions of the eyes, and hearing not profoundly affected, present a positive rotation, caloric and fistula tests, one would reasonably expect to find at operation a fistula in some part of the labyrinthine capsule and would no doubt be able to find it.

Prognosis.—A fistula may heal after the cause producing it has been removed or it may result in a serous or purulent labyrinthitis. This may occur either before or after operation, for the middle-ear process of which it was a complication.

Treatment.—These fistulæ, when found at operation, are to be let alone. A sequestrum, if present, may be removed. If granulations are exuberant, one may no doubt justify oneself if he carefully curettes them. However, the barriers between the suppuration and the healthy labyrinth must be conserved; therefore no meddlesome probing or other manipulation is justifiable, nor can a primary graft be used. After operation the patient is to be watched carefully for extension of the process to the hitherto uninvolved labyrinth, that proper measures may be promptly undertaken should it occur.

SEROUS LABYRINTHITIS.

Serous labyrinthitis may result from the circumscribed form—secondary serous labyrinthitis—or it may follow

the radical operation or suppurative middle-ear processes —induced serous labyrinthitis.

Pathology.—A suppurative process either at the site of a fistula or in the recesses of the oval or round windows seems sufficient to cause a serous or serofibrinous labyrinthitis. The bacteria proliferating at the oval and round windows cause swelling and infiltration of the tissues with a fibrinous deposit on the internal surface of the membrana tympani secondaria and the annular ligament of the stapes. There is a dilatation of the labyrinthine bloodvessels and increase in the amount of perilymph. The pressure caused by increase of perilymph produces various distortions, and adhesions may form making resolution with restoration to normal function impossible. The utricle and saccule may be compressed and the ductus cochlearis distorted. This tube may be collapsed at the apex and dilated at the basal whorl or from increase in endolymph dilated throughout. These changes in the cochlear duct or scala media, containing as it does the organ of Corti, naturally result in profound impairment of hearing. Round cells are found in the labyrinth but no bacteria; once these appear on the inner surface of the structures closing the oval and round windows the disease very rapidly becomes purulent. In not all patients are the changes in structure so profound, for return of hearing at times occurs to nearly the condition present before the development of the disease.

Symptoms.—The onset of the symptoms is usually quite sudden. The patient experiences marked vertigo accompanied by nausea and vomiting. There is nystagmus to the side away from the disease. This may be proceeded by nystagmus to the diseased side, but this is not often observed, as it usually disappears with the onset of the symptoms of "destruction disharmony"—vertigo, nausea and vomiting, nystagmus to the healthy side. These are brought about by the sudden cutting

out of the impulses which normally emanated from the involved labyrinth. The labyrinth ceases to functionate and an imbalance or disharmony is produced. One could understand this better if he assumed that the imbalance was between the two labyrinths alone; but experiments have demonstrated that the condition is by no means so simple, as these phenomena take place in animals after destroying the one remaining labyrinth, the first one having been previously destroyed and the symptoms of the destruction disharmony having been recovered from. So when the labyrinth, from a serous or purulent inflammation, ceases to functionate, its impulses are cut off from the intricate nervous mechanism which regulates equilibrium and orientation and the symptoms of destruction disharmony result. There is regularly impairment of hearing which tests show to be due to a lesion of the perceptive mechanism. It varies with the severity of the process, at times deafness becoming absolute. The caloric and rotation tests may or may not be positive. The nystagmus to the opposite side, besides the other symptoms of destruction disharmony, is caused by some interference with the movement of the endolymph within the labyrinth. As soon as this is sufficient to produce an imbalance nystagmus results. The endolymph may still be capable of movement, although it does not move with sufficient freedom to stimulate the cristæ ampullæ as in a normal labyrinth. The currents produced by the caloric, rotation and fistula tests, being much more powerful than the physiological currents in the fluid, may still be capable of so stimulating the cristæ that the involved labyrinth becomes the predominating one in producing an imbalance and hence the tests be positive.

Diagnosis.—The diagnosis is made upon the presence of the vertigo and nystagmus to the opposite side in conjunction with the result of the tests of hearing and the irritability of the static labyrinth. The following

table from Ruttin shows the tests in the various stages of labyrinthitis:

	1	2	3	4	5
Hearing . . .	+	−	−	−	−
Caloric . . .	+	+	−	−	−
Rotation . . .	+	+	+	−	−
Fistula . . .	+	+	+	+	−

The four tests are arranged according to their delicacy and in the order in which they are usually lost. As long as the reactions are represented by the first four columns the process may be considered a serous one. When all of the reactions are lost as in column 5 it may still be serous but there are no means yet known to determine that such is the case. If after a week or so the reactions begin to return, then it is fair to assume that the process has been a serous one all the time and that it is now resolving. However, during the absence of the reactions it so closely resembles a purulent labyrinthitis that there is no difference in the clinical picture of the two diseases.

Prognosis.—The disease may pass into the purulent type of labyrinthitis or may result in cure with more or less impairment of hearing. In mild types this may return to practically as good as before, while in the severe types total deafness may result. The vertigo usually passes away in from three to ten days, although a mild degree may persist for some time. This is also true of the nystagmus, although at times it seems slow to disappear. The author has under observation a patient in whom the nystagmus to the opposite side is still present some four months after the attack of serous labyrinthitis.

Treatment.—As long as the labyrinthitis is of the serous type, there are no bacteria in the labyrinthine cavity and there is no danger of meningitis; therefore operation is not indicated. Moreover, it is not unreasonable to expect the patient to get well with some return of function. The only reason which would justify operation is that the diagnosis between serous and purulent labyrinthitis could not be made, and one would then

be operating upon the assumption that the disease was no longer serous in nature. If the reactions have been gradually lost, one at a time during a few days, one would be justified in postponing operation, although this history may occasionally be given in purulent processes. So the patient is treated expectantly with rest in bed, light diet, and laxatives.

PURULENT LABYRINTHITIS.

Definition.—Suppurative labyrinthitis is either manifest or latent. The term manifest is applied to those processes in which the symptoms of destruction disharmony are present. As nature accustoms herself to the loss of the impulses normally derived from the destroyed labyrinth the symptoms disappear, and the process becomes latent. Whether it is possible for the destruction of the labyrinth to take place so gradually in this disease that the symptoms of the disharmony may never be present is undecided. It must, however, be of rare occurrence, although patients occasionally present in the latent stage who give no history of the symptoms of the manifest stage.

Causation.—The disease is an infective process involving the whole labyrinth. Infection may occur through the oval and round windows, through a fistula in the promontory or any part of the labyrinthine capsule, or it may be secondary to a purulent meningitis, or be due to septic embolism of the internal auditory artery, although usually the process follows directly upon middle-ear disease. Penetrating wounds of the labyrinth as by the introduction of a foreign body or by the instruments of the surgeon, are usually followed by purulent inflammation. A myringotomy knife entering the oval window, a defect in the annular ligament of the stapes caused by traumatism inflicted upon this ossicle during operation, or the accidental extraction of the stapes in an infected field, are each sufficient to produce a suppurative laby-

rinthitis, or the disease may follow operations in which no known traumatism upon the labyrinth has been produced.

Pathology.—The membranous labyrinth becomes inflamed and infiltrated with round cells. The delicate membranes in the cochlea, especially Reissner's, rupture and the endo- and perilymph spaces become filled with purulent material. An abscess may develop in the internal auditory meatus which may be walled off from the general meninges by adhesions. This abscess may cause destruction of the internal auditory artery which supplies a considerable part of the bony labyrinth, and this may lead to necrosis of the whole or part of the labyrinth. The infection may extend to the meninges through the internal auditory meatus, through the aqueductus cochleæ, or more rarely through the aqueductus vestibuli. The infection in the latter may result in empyema of the ductus endolymphaticus or an interdural abscess which may in turn lead to cerebellar abscess. There is regularly degeneration of nerve tissue within the labyrinth, although at times it seems quite resistant. As the process goes on, there may be in some instances formation of fibrous tissue or new bone so that the labyrinthine cavity becomes completely filled with this tissue. If this exists the process may be considered as healed. As restoration of function never takes place, the most that can be hoped for is healing with the bone or fibrous tissue, in which event it may be considered probable that the menace to life from complications is at an end.

Pan Otitis.—Many years ago Politzer described a form of purulent labyrinthitis occurring in acute otitis media under the name Pan Otitis. The middle ear and labyrinth were apparently simultaneously involved. The disease occurred most frequently in children, upon one side only or upon both sides. There was a febrile attack of greater or less severity accompanied by vertigo and vomiting. When the patient recovered, profound or

total deafness was present. Examination of the membrana tympani, after recovery, showed evidences of middle-ear suppuration, although in some instances these could with difficulty be made out.

Symptoms.—In the manifest form there is deafness, vertigo, nausea and vomiting, nystagmus to the healthy side, and if the patient tries to walk, a staggering gait. The patient usually takes to bed and lies upon his well side. This decubitus is quite characteristic and also occurs in serous labyrinthitis. It is due to the patient's subjective sensation of rotation which is part of his vertigo. He finds this increased when he looks in the direction of the rapid component of his nystagmus and less when he looks in the direction of the slow component. So he lies upon his well side and is thereby enabled to look in the direction of the slow component of his nystagmus which is toward the diseased side, since the rapid component is toward the good side, as above stated. If he adopts any other position he usually keeps his eyes closed. There may be some fever but only of a degree or so. Fully as often the disease is afebrile. This is not unreasonable when one considers how small the labyrinthine space is and that the absorption of the products of inflammation which produce fever is not so free from the inside of a bony cavity. If there is a rise of more than one degree in the temperature, the occurrence of some intracranial complication, probably meningitis, should be suspected. Severe pain ought also to suggest meningitis. If the labyrinth is tested during this manifest stage, it will be found to give no response to any of the four tests. After a varying time, from three to nine days, the vertigo disappears. It is usually mild after the first three days. The author had one patient in whom it lasted but twenty-four hours. The nystagmus, however, is usually slower in disappearing but it usually becomes of milder degree in from one to three weeks. When it becomes nearly absent the patient may be considered to be in the latent stage. The terms

manifest and latent are merely used for convenience. There is no exact dividing line between them. When the patient has no symptoms referable to his labyrinth except deafness or tinnitus he is usually considered to be in the latent stage. He still remains negative to the four tests and if he comes under observation during this stage, these tests, the evidences of a suppurative ear process and the history of the manifest stage usually are present. If he does not give a history of vertigo, one may be in doubt but these instances are not common. If upon rotation it is found that compensation has occurred, it is fair to assume that the process within the labyrinth is a healed one.

Sequestration.—In the latent stage occur the results of those processes which produces death of a whole or part of the labyrinthine capsule. The sequestrum may be extruded and discharge with the aural pus. In this case it is usually some small part of the cochlea. Or it may be found lying in a bed of granulations at the operation. Outside of the tests usually present in the latent stage, facial paralysis is often present in sequestration. It is difficult for a sequestrum of any size to form which is composed of labyrinthine capsule, without at the same time affecting the Fallopian canal, so the occurrence of facial paralysis in a patient with diffuse suppurative labyrinthitis should always lead one to suspect sequestration.

Prognosis.—The prognosis of suppurative labyrinthitis is bad. While many patients may have the condition for years and in some it becomes a healed process, the danger of meningitis and cerebellar abscess is so great that it must be considered a very serious condition.

Treatment.—Outside of the usual measures adopted in any inflammation in the temporal bone, the treatment of suppurative labyrinthitis is a subject fraught with difficulties. Some surgeons are so "conservative" that they only operate when meningitis is "imminent," being mainly guided to this conclusion by rise in temperature

or headache. If the operation is done then, the meningitis has already begun and it is too late in most instances, although a patient is occasionally saved. If death occurs under these circumstances, it cannot be attributed to the operation. Then, again, some patients get well; that is, apparently so, although with loss of function, without operation. The proportion that do this is by no means negligible. Nevertheless there can be no doubt but that a patient with a suppurating labyrinth is better off if it is drained. The mortality would certainly be less if every such labyrinth was opened by a skilful operator thoroughly equipped by dead-house work to perform the operation. Before the radical operation every patient should have his reactions taken and if a latent suppurative process is present, the labyrinth should be drained at the time of the radical operation or the latter not performed. An exception to this rule may be made when compensation has occurred—indicating a healed process. When there is latent diffuse labyrinthitis with facial paralysis, the radical operation should be done, search made for a sequestrum and, if found, removed.

PERILABYRINTHITIS.

Definition.—The term perilabyrinthitis is applied to a certain variety of clinical manifestation that cannot with consistency be called by any of the other names used to designate the processes complicating middle-ear suppuration. It is probable that as the knowledge of labyrinthine conditions become more complete, the term will become obsolete, as there are many objections to its use.

Pathology.—The lesion is supposed to be an inflammation of the bone contiguous to the labyrinthine capsule involving to a certain extent the capsule itself. As the processes coming under this head are not fatal unless they pass into some of the other forms of labyrinthitis, reports of autopsies in patients afflicted with this con-

dition are not forthcoming and the exact nature of the basis of the disease is more or less a matter of speculation. It occurs after the radical operation, less often after simple mastoidectomy and as a complication of a suppurative process in the middle ear and mastoid.

Symptoms.—The patient usually comes on account of the vertigo which he experiences. This is not the profound dizziness of manifest suppurative or serous labyrinthitis but is milder. Some patients complain of more or less continuous vertigo which does not compel them to go to bed as a rule, but may render them unfit to pursue their vocations. It is not accompanied with vomiting except in rare instances. This dizziness is aggravated or brought on by sudden movements of the head. In other patients the vertiginous symptoms come in attacks, while between the attacks they are more or less free. If these patients are made to hop forward or backward with the eyes closed, they usually, but not constantly, show a tendency to fall, but the author has been unable to note that there is any constant relation between the direction of the tendency to fall and the side supposed to be involved. There is regularly nystagmus, usually of mild degree, seen in the extreme lateral position of the eyes and either to the involved side or to both sides.

The hearing is not affected or but moderately so. The caloric and rotation tests show a normal or increased irritability of the labyrinth and the fistula symptom is negative.

Diagnosis.—The diagnosis is to be made by excluding the other forms of labyrinthine disease. The only one which it resembles is circumscribed labyrinthitis. From this it is separated by the increased irritability to the caloric and rotation tests which is usually present and by absence of the fistula symptom.

Prognosis.—The prognosis is usually good, although a patient having this form of disease may occasionally develop one of the other and more dangerous types of labyrinthitis.

Treatment.—When the condition follows the radical or simple mastoid operation, the treatment is largely expectant. Full doses of the bromides are at times of benefit. Pilocarpine may also be given and at times seems to be of some value. The patient is supplied with a 2 per cent. solution of the hydrochlorate and directed to take two drops at night for three days, then if the physiological action of the drug has not manifested itself by sweating or flow of saliva, the dose is increased by one drop every three days until these symptoms do occur. The drug should be watched carefully, as at times it makes the patient feel weak instead of producing sweating or flow of saliva, indicating that the dose is too large. When perilabyrinthitis occurs on account of a chronic suppuration or an acute mastoiditis, an operation is indicated to remove, if possible, the involved area adjacent to the labyrinthine capsule.

LABYRINTH OPERATIONS.

Choice of Operations.—Labyrinth operations may be divided into two classes: (1) Those which have as their object opening and draining the labyrinth; (2) those which while accomplishing this are continued further and the internal auditory meatus opened and the dura in the middle and posterior fossæ exposed. The first are to be performed when the infective process is still confined to the labyrinth, and the second when invasion of the meninges has occurred or seems imminent. As has been seen in the preceding pages, the indications for labyrinth operations are still somewhat indefinite; but granting that an operation is necessary in a given patient, the decision as to the procedure to be adopted largely rests on the intracranial condition. If an abscess has formed in the internal auditory meatus, or if there is a meningitis on the posterior surface of the petrous pyramid, or if there is a serous meningitis due to a labyrinthine suppuration, labyrinthine drainage would be futile, as it

would also in a general meningitis from infection through the labyrinth. On the other hand, when the process is limited to the spaces within the labyrinth or one feels reasonably sure that this is the case, one would hesitate before subjecting a patient with such a process to the more severe surgical procedure, but would be content with labyrinthine drainage.

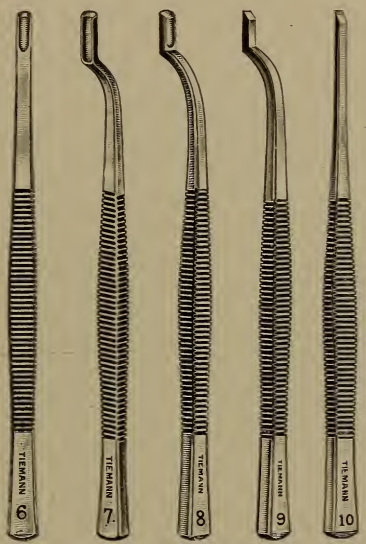

FIG. 110.—Author's set of labyrinth gouges and chisels.

Preliminary Operation.—These operations can only be performed after the radical operation, so if it becomes necessary to do one in a patient who has not previously had this operation performed, it must be done as a preliminary procedure. A very thorough operation is necessary, the facial spur must be taken down to its extreme limit, all cells and soft tissue removed so that the white ivory-like bone of the labyrinthine capsule shows to as great an extent as possible. The promontory of the cochlea and the horizontal semicircular canal show plainly

in the radical operation, but it may be possible after the soft bone is removed to see the posterior and part of the superior canals also. The operator must be thoroughly acquainted with the anatomy of the parts, in fact, possess such a knowledge as can only be derived from a frequent performance of the operation on the cadaver. The operation then becomes one of no great difficulty and adds very little to the mortality even if it is not the means of saving the life of the patient. It is because the operation is deferred until the process demanding it is practically hopeless that the mortality is so high. Anyone competent to do a radical operation, if he will take the pains and time necessary, can become able to perform the labyrinth operation with skill and safety.

Instruments.—The instruments necessary are those with which the radical operation is done, a set of gouges and chisels (Fig. 110) and a very fine probe.

LABYRINTHINE DRAINAGE.

This procedure is termed labyrinthine drainage, as the prime objects of the operation are to open the vestibule and cochlea, thus affording thorough drainage to a purulent process within the labyrinth. This much can be accomplished in any type of bone by the procedure about to be described. In many instances the whole canal system can be removed if thought advisable, but if there is a low middle fossa with a far forward sinus the technical difficulties encountered in trying to accomplish this may be very great.

Guide to the Vestibule.—The vestibule is to be opened first, the guide to this cavity is the horizontal semicircular canal, the anterior or ampullated extremity of which opens into the vestibule at its anterior and upper part, while the posterior end opens into this cavity at its posterior and upper part. It is evident, therefore, that the bone between these two ends is the roof of the vestibule,

and it is this roof which it is proposed to take away, thereby opening and draining this cavity.

Opening the Horizontal Semicircular Canal.—The first step is to open the horizontal semicircular canal and trace its lumen from end to end. The anterior half or two-thirds of this canal is immediately above the facial nerve. In this situation the upper wall of the Fallopian canal and

FIG. 111.—Labyrinthine drainage. Horizontal semicircular canal opened.

the lower wall of the semicircular canal are one and the same process of bone. This lies over the oval window and the osseous semicircular canal is the real support of the facial canal in this location, so in opening the lumen of the anterior part of the horizontal canal and in the further progress of the operation, all of the inferior and as much as possible of the external wall should be

preserved. So with a bent gouge a pit is started above and slightly internal to the concavity of the horizontal semicircular canal. Working outward, shaving after shaving is taken until the canal is opened. This opening is then extended until the two ends of the canal are reached (Fig. 111). A fine probe can now be passed into the vestibule through either end of the canal.

Opening the Vestibule.—This locates the vestibule and the bone between the two ends of the horizontal canal must be removed. This can be done with the two bent gouges, with the one working from behind forward and with the other from before backward, deepening the pit already started, and taking care not to encroach on the floor of the semicircular canal in its anterior half or two-thirds. If there is room between the descending part of the facial nerve and the posterior cranial fossa, a gutter may be formed in the angle between the posterior semicircular canal and the posterior end of the horizontal canal. This is termed the hard angle, and the gutter formed in this angle may remove the posterior end of the horizontal canal and part of the posterior canal also. This will afford more room to remove the roof of the vestibule. The operator must keep the two ends of the horizontal canal always in view and not allow them to become filled with débris and thus obscured. At times in completing the opening in the vestibule a very narrow chisel is useful.

After the vestibule is opened, if the operator desires to remove the posterior and superior semicircular canals, he proceeds to do so. In removing the superior canal in its entirety it is necessary to expose the dura and remove the inner table to a point internal to the eminentia arcuata. The anterior end is opened with the bent gouges which are also very useful for opening and tracing the posterior canal.

Opening the Cochlea.—The next step is removing the internal tympanic wall over the cochlear whorls. The first turn is opened by removing the promontory and

the process of bone between the oval and round windows. This is best done with a small gouge. The cutting edge is placed upon the promontory immediately below the oval window, and a shaving taken in a downward direction extending to near the floor of the tympanum. The instrument is pointing directly toward the jugular bulb in this situation and must be under perfect control. The shavings taken must be thin, as if thick ones are taken the gouge may jam into the cavity of the cochlea and fracture the modiolus. This accident, as shown by Richards, not only allows the escape of cerebrospinal fluid but also opens an avenue through which infection may extend to the meninges. The fluid drains through the openings caused by tearing the meningeal sheaths which accompany the nerve fibers to a certain distance after they enter the modiolus. After the promontory is removed there is usually left a triangular piece of bone between the oval and round windows, the third side being formed by the margin of the cut in the promontory which has been made with the gouge. A curette may be carefully inserted, just taking this edge and by withdrawing it the bone is drawn out with safety. This completes the opening of the first cochlear turn and, unless it is determined to open the second turn, the operation also.

Facial Nerve.—A bent probe can now be passed from the cavity made while opening the vestibule through the cochlear opening into the lower part of the tympanic cavity (Fig. 112). In this position it lies beneath a bridge which has been formed by the operation. This consists of the Fallopian canal reinforced by the lower, and as much as has been left of the external wall of the horizontal semicircular canal. This bridge should always be left, as to remove it produces permanent facial paralysis and does not add materially to the drainage. Even though it should be found partly necrotic as it may be in some instances of labyrinthine sequestration, an attempt should be made to leave as much of it as possible, as no substitute can be found for even a small part of the

Fallopian canal. This is the best structure to support the nerve and thus minimize facial paralysis.

Second Cochlear Whorl.—If it is determined to open the second turn of the cochlea, this can be done with the gouge. The bone covering it appears pearly white upon the internal tympanic wall anterior to the opening made

FIG. 112.—Labyrinthine drainage. Vestibule opened posteriorly and above; promontory of the cochlea removed. Probe passes beneath the bridge containing the facial nerve.

by removal of the promontory. Thin shavings are taken from this surface until it is opened. The carotid artery lies in front, the jugular bulb below and the facial nerve above the area to be removed; so considerable care is necessary to avoid these structures. While it is a question how much additional drainage is afforded by opening this turn, the author has always felt when doing it on the

living, that it was more an exhibition of operative possibilities than a proceeding of decided value.

After-treatment.—The after-treatment of this labyrinth operation is very much the same as in the radical operation. No primary graft is applied. If the patient does well, a secondary graft may be inserted in two or three weeks, when the posterior wound may also be closed, as this is usually left open temporarily after this procedure.

Neumann's Operation.—Of the second type of operations on the labyrinth, that introduced by Neumann is the one usually done.

Locating the Vestibule.—The first step is exposing the lateral sinus. This is done in the same manner as in sinus thrombosis (which see). After the internal border of the sinus is exposed the bone is removed in Trautman's triangle either with the rongeur, curette or, as Neumann advises, with a chisel. The dura is to be separated from the bone and with the chisel pieces are taken from the posterior surface of the petrous pyramid. These shavings are very apt to jam between the dura and the bone and must be removed as they are formed. The author has found the curette a very useful instrument in this step of the procedure. It can be introduced between the dura and inner table and by using the posterior edge of the bone wound as a fulcrum the pieces taken are removed with expedition and safety. This is continued until the posterior semicircular canal is reached. This is opened and its prominence removed. A fine probe will now pass through either opening into the vestibule. The lower one enters through the ampullated extremity of the canal into the lower part of the vestibule while the upper enters through the crus commune to the upper and back part of this cavity. These two openings are therefore the guides to the vestibule.

Opening the Vestibule.—As the removal of the bone shavings from the posterior surface of the petrous pyramid is continued a third opening develops between the other two. This is the posterior end of the horizontal canal

and furnishes an additional guide to the vestibule (Fig. 113). This cavity is soon opened. When it is freely exposed one may be working very near the descending limb of the facial nerve, the location of which may be determined by estimating how much of the posterior end of the horizontal canal has been removed, as the nerve turns downward about in the middle of this canal or at the junction of its middle and posterior third.

Fig. 113.—Neumann's labyrinth operation, showing the two openings of the posterior semicircular canal and more anteriorly (to the left) the posterior end of the horizontal semicircular canal.

Opening the Internal Auditory Meatus.—The next step is the removal of the triangular process of bone which lies between the vestibule and the internal auditory meatus. This meatus ends at the vestibule, as the vestibular nerves occupy its posterior part and some of them pass directly into the vestibule, which thus becomes the guide to the internal end of the meatus. With the curette, small gouges and chisels the triangular piece of bone is removed, taking care that the dura is separated from the bone. Neumann advises that the dura in the middle

fossa be exposed but the author prefers to leave the inner table in this location until the end of the operation for two reasons: (1) There is more danger of wounding the superior petrosal sinus if the angle of bone between the posterior and middle fossæ is taken out. This has occurred and the bleeding is a serious matter, as the field becomes completely obscured; (2) this process supports the bone upon which one is working and prevents fracture in some untoward locality. That this danger is real is shown by

FIG. 114.—Neumann's labyrinth operation. The internal auditory meatus opened but not incised.

the sensation of instability communicated to one's hand at each stroke of the mallet. As removal of this triangular piece proceeds a bent probe passed between the dura and posterior surface of the petrous pyramid will soon slightly enter the internal auditory meatus. Thus both ends of the meatus being established, it only remains to remove the bone between them, when the dura lining the internal auditory canal is exposed (Fig. 114). This is opened by a longitudinal incision. If an abscess is found, no further dural incision is to be made unless meningitis is known to

be present. If an abscess is not discovered, the incision should be continued into the cerebellar dura. The dura in the middle fossa may now be incised, the inner table being removed if it has not already been done.

Cochlear Drainage.—Before these dural incisions are made it is usually a good plan to open the cochlea. This is done in exactly the same manner as described under Labyrinthine Drainage.

After-treatment.—In some patients the flow of cerebrospinal fluid is quite free for from twenty-four to forty-eight hours, when it generally ceases from the formation of adhesions. In others it is scanty or absent. The dressings are changed daily or every second day and the wound heals by granulation and dermatization as any radical cavity.

CHAPTER XI.

COMPLICATIONS OF PURULENT OTITIS MEDIA.

MENINGITIS.

Meningitis as seen by the otologist may be discussed under four heads: (1) External Pachymeningitis. (2) Circumscribed Meningitis. (3) Purulent Leptomeningitis. (4) Serous Meningitis.

External Pachymeningitis. — External pachymeningitis as usually seen is the ordinary extradural abscess. As the disease in the mastoid progresses the internal table breaks down and the pus accumulates between the dura and the bone. In some instances one feels inclined to believe that this has taken place, being led to this conclusion by the sudden cessation of severe pain or diminution of the discharge. If pain in a given process is caused by pressure of pus within the mastoid, rupture of the inner table removes the tension and relieves the pain and may also cause a diminution of the amount of the pus which appears in the meatus. The dura reacts to this purulent irritation and granulations of varying size form upon its external surface. It is not an infrequent occurrence to see granulations as thick as one's finger during operation upon patients, in whom the process has been going on for some time and has been neglected. The treatment is considered under the mastoid operation.

In another type the internal table is softened over a very small area or the infection has extended through without breaking down the bone. There is a purulent inflammation of the external dural layers and the pus

accumulates between this membrane and the bone, exceptionally in sufficient quantity to cause increased intracranial pressure and, if in proper position, the localizing symptoms of brain abscess.

Diagnosis.—The diagnosis of these processes may be difficult. One may be led by the symptoms to suspect some pathological intracranial condition and upon exposing the dura discover the abscess. One symptom which should never be neglected is the sudden discharge of a quantity of pus. A mastoid operation may have been performed and the wound apparently healing normally when, upon removing the packing, pus will be present in varying quantity. Bacteriological examination of this pus will furnish some information. The germs causing a mastoiditis usually soon disappear after operation, as smears taken from the wounds soon show that the prevailing germ cannot be found; so if this germ is present in this purulent secretion which appears in a wound previously normal, its source should be investigated. It may be absent for a dressing or two, then reappear. One may at times be able to detect the location from which the discharge comes or the probe may reveal the opening or necrotic bone in its vicinity. Politzer was able to demonstrate the presence of such an abscess in the middle fossa by means of the otoscope. Upon rarefying the air in the meatus pus was drawn through a small perforation in the tegmen tympani. One should think of this form of meningitis when trying to ascertain the cause of headache, especially if localized about the ear.

Treatment.—The treatment is to open and drain the abscess, removing sufficient bone to accomplish this, and is to be carried out along the lines described under the simple mastoid operation.

Circumscribed Meningitis.—Occasionally the internal surface of the dura and the pia arachnoid become inflamed and instead of spreading the process becomes circumscribed. If the condition progresses, the external surface of the brain breaks down and an abscess is formed. Cases

of circumscribed meningitis with ulceration of the brain are usually reported as "brain abscess." The surgeon incises the dura and pus is discharged which contains broken-down brain tissue, the abscess really lying within the meninges (subdural) in contradistinction to true brain abscess which is located within the brain substance.

Causation.—The process is usually caused by contiguous extradural abscess, either infection of the internal surface of the dura and pia arachnoid, in which event the localized inflammation is purulent, or swelling and edema of the dura results in the effusion of serum within the subdural space, which becomes circumscribed by adhesions. A sinus thrombosis in which the clot breaks down may be the cause, the meninges adjacent to the internal sinus wall becoming involved.

Symptoms.—The symptoms in the early stages are very much like those of general meningitis but not as severe. In the later stages they very closely resemble those of brain abscess. In the serous type there may be no symptoms, the surgeon on account of the tense character and perhaps also some discoloration of the dura, being led to suspect the presence of an accumulation of fluid, incises the membrane, evacuating the fluid, or there may be pain and increase of temperature, as in the early stage of the purulent process.

Diagnosis.—In diagnosing these processes one has the findings of the mastoid or radical operation as a start. If some condition has been found that would conduce to meningeal inflammation and the symptoms of meningitis develop and upon examination of the spinal fluid it is found to be not far from normal, one comes to the conclusion that the process is localized. It may be impossible to separate the process from brain abscess, in fact the difference may not be discovered until operation or not even then, the two conditions resembling each other so closely in some instances.

Prognosis.—These circumscribed processes, if recognized and treated surgically, offer a good prospect of cure,

although at times they may eventuate in general meningitis.

Treatment.—The treatment is to afford free drainage by incising the dura. In making this opening care should be taken not to extend it beyond the limits of the circumscribing adhesions, at the same time it must be sufficiently free to afford drainage. A cigarette drain or folded rubber tissue may be inserted to keep the dural wound open. No examination of these cavities either with the finger or any instrument is to be allowed, as nature's barrier adhesions may be broken down and the process thus become general.

Purulent Leptomeningitis. — Purulent meningitis of otitic origin is the *bête noir* of the otologist. The possibility of its occurrence makes every case of suppurative middle-ear disease a serious matter.

Causation.—It may be caused by acute or chronic suppuration in the middle ear or mastoid or be due to rupture of a brain abscess either into the ventricles or upon the surface of the brain. The infection may reach the meninges through a wound of the dura, produced during operation, or from an infected sinus or extension of an extradural abscess. Solution of continuity of the dura is not necessary, as the infection may extend by a clot in the small veins or through the lymphatics. Infection may be transmitted in this manner through the vessels and lymphatics which exist between the tympanic vault and the middle fossa or there may be a dehiscence in this location, the mucous membrane in the middle ear, on account of this defect, coming into very intimate relation with the dura. Instances are on record in which the meningitis was caused by evulsion of a polyp which had its attachment to the soft structures in such a dehiscence. The infective process may travel along the nerves. In this manner involvement within the Fallopian canal may cause meningitis, the infection reaching the meninges either through the hiatus Fallopii or the internal auditory meatus. The petrosal branches of the tympanic plexus

also perforate the tegmen tympani and may form the avenue of infection. Wittmaak[1] believes that bacteria may find their way from the hypotympanum to the meninges through the small vessels and along the nerve sheath of the tympanic branch of the glossopharyngeal, thus gaining entrance to the process of the meninges which surrounds the ninth, tenth and eleventh cranial nerves in the jugular foramen. Of special importance as causing meningitis is suppurative disease of the labyrinth. The usual route of extension is through the internal auditory meatus, next in frequency the aqueductus cochleæ, and less often the aqueductus vestibuli. At the internal auditory meatus the circulatory arrangement seems very favorable for the transmission of infection from the labyrinth to the meninges. Practically the entire blood supply of the labyrinth passes through the internal auditory meatus; moreover, at the fundus of this canal the thin shell of bone is perforated to transmit the vessels and nerves which in some locations are covered with meningeal processes even after their passage through the foramina. Attention has already been called to the effort on the part of nature to limit the infection in this canal and the consequent production of an abscess. If not relieved promptly these abscesses rupture, leading to meningitis.

Pathology.—In purulent leptomeningitis there is injection of the pial vessels, formation of a purulent exudate, softening of the cortex and more or less edema of the brain substance. There is increase of the cerebrospinal fluid which is first clear, afterward cloudy or purulent. The ventricles may be distended and intracranial pressure increased. The fluid also collects in increased amount in the various cisterna of the pia arachnoid. The process also extends to the spinal meninges. There is absorption of the products of inflammation and symptoms caused by loss of function of the brain cortex, especially at the base.

[1] Zeitschrift f. Ohrenheilk., vol. xlvii.

Course and Symptoms.—A purulent meningitis may run an acute course, death taking place in twenty-four to forty-eight hours. This very acute or fulminating type is apt to follow rupture of a brain abscess into the ventricles. More often the course is slower and death does not occur for a week or so, while in some patients the disease seems to be more chronic or even latent for short periods of time. Sometimes one of these latent types is lighted up into an acute form by the performance of the radical operation. Alexander advises a lumbar puncture at the time of operation, if any suspicious symptoms are present, to ascertain whether or not the meningitis is due to operative interference, and T. Passmore Berens goes a step further and proposes to do a lumbar puncture even in the absence of symptoms, being influenced in this decision by the observation of several patients with the latent form of meningitis. The most common symptom is headache, usually coming on early and remaining more or less continuous. It may be referred to the frontal or occipital regions or to any part of the head. Sometimes it is more severe in the neck, radiating to the head. Rarely it is absent or not complained of, especially in latent types. Vomiting is also common either as a result of a manifest purulent labyrinthitis or as a symptom of the meningitis itself. There is regularly a rise of temperature. This may not be high but usually varies between 101° and 105° F. In the beginning the pulse is rapid with usually an increased arterial tension. After the development of increased intracranial pressure, it usually becomes slower but not slower than normal. There is increase of the reflexes and the patient is easily startled.

The Kernig sign is usually positive, although this may occur in other conditions also. In a normal person the thigh may be flexed upon the abdomen to a right angle or even beyond without bending the knee. If the Kernig sign is positive it will be impossible to bring it to a right angle with the trunk without bending the knee. The

Babinski sign may also be positive. If the sole of the foot of the normal person is lightly stroked, there will result flexion of the toes, especially of the great toe. If the Babinski is positive, stroking the sole will produce extension dorsally.

There may be inequality of the pupils or they may not react normally. There may be various paralyses of the eye muscles. Abducens paralysis is present in about 3 per cent. of the cases. The fundus is very apt to show changes. In one hundred cases analyzed by the author, some change was noted in about one-third, in one-third it was absent, while in the remaining third the condition of the eye-grounds were not mentioned. Yansen states that some change is present in about 50 per cent. of the patients.

If the disease follows a suppurative labyrinthitis, there may or may not be nystagmus, but there is loss of the reactions as characteristic of that disease. Stiffness of the neck is a very common symptom, in some instances the rigidity extends the entire length of the back, resulting in opisthotonos. As the disease progresses the patient passes into a semiconscious state and finally cannot be aroused at all. He cries out at times with a sharp moan that becomes truly distressing to hear.

Diagnosis.—In making a diagnosis great reliance is placed upon the result of lumbar puncture. The fluid may or may not be under abnormal pressure, but the evidence afforded by its examination is of great value. A smear may be made and bacteria found but in the early stages a culture is necessary to determine their presence. Bacteria in the spinal fluid establishes the diagnosis of purulent meningitis. If the variety of germ is the same as found in the mastoid pus, it is a fair inference that the infection is from this location; if not, further search must be made to determine its source, which in some instances will be disease of the accessory sinuses of the nose. If bacteria cannot be demonstrated by culture, it does not necessarily follow that the patient does not have this

disease. The author treated a patient who had purulent meningitis in whom repeated cultures failed to demonstrate the presence of any pathogenic microörganism in the spinal fluid, which was in other respects characteristic. The patient became steadily worse and died in about twelve days from the onset of symptoms. It was suspected that an anaërobic bacteria might have been present or that the meningitis was tubercular in nature. An attempt was made to ascertain the presence of tubercular bacilli by inoculating a guinea-pig but without result. In another patient, who ran a fatal course in about four weeks, it was only upon the eighth lumbar puncture with cultivation of the fluid that a pneumococcus was demonstrated, the character of the fluid as well as the clinical manifestations, in the meantime, leaving no doubt as to the nature of the process. Before the culture is positive or to make a diagnosis and adopt treatment without waiting the necessary (twenty-four hours usually) time for a report of its result, certain characteristics of the fluid may be of value.

The cerebrospinal fluid normally contains a small amount of sugar and readily reduces copper by the Fehling test. It has been found that the growth of bacteria within the meninges may exhaust this sugar content. Thus absence of sugar becomes a sign of some value. It is difficult to estimate its true importance, but a disposition to consider it of small worth seems to be growing. Taken in conjunction with the leukocyte count, which is of far more importance, failure to reduce copper on the part of the fluid should have some weight in pointing to purulent meningitis.

Normal cerebrospinal fluid does not contain more than ten leukocytes to the cubic millimeter. It is claimed by some to contain even less. Moreover, these cells are of the mononuclear variety. An increase in the number of cells and the occurrence of a substantial polynuclear percentage is strong evidence of meningitis, although this may occur in cerebral syphilis, perhaps other conditions

also. In acute purulent meningitis that has once shown sufficient symptoms to make itself suspected there is likely to be very little chance of error, although many problems relating to the cerebrospinal fluid in disease of the central nervous system remain to be solved. A fluid in a suspected meningitis will usually contain sufficient leukocytes of the polynuclear variety to either confirm one's suspicion or show that it is without foundation. It is impossible to state a number below which the patient does not have and above which he does have a purulent meningitis. The author has known the first lumbar puncture, in a fatal case of purulent meningitis, to give fluid with a leukocyte count below 100 per millimeter, but this is by no means the lower limit. On the other hand, a puncture made late in the disease may draw fluid with a leukocyte count in the thousands. Nor can one make a diagnosis between purulent and serous meningitis on the leukocyte count alone, although a certain amount of evidence is afforded. The count should be low in serous meningitis but high in purulent meningitis. The author believed that the count should be under 60 in the serous process, but a recent experience has taught him that this is placing it too low.

A patient with purulent labyrinthitis on the day following the onset of the disease had a temperature of 101.6° F. but no headache. A lumbar puncture showed the fluid under pressure. Fehlings +, globulins +, leukocytes 145, polynuclears 70 per cent. The labyrinth was removed and the internal auditory meatus opened within four hours of the report above given. After twenty-four hours the culture was negative. The fluid drained from the wound for four days, the patient having nocturnal headache, some stiffness of the neck and a positive Kernig, the temperature varying between 99.8° and 102.6° F. The spinal fluid would not flow on lumbar puncture. This was attributed to the fact that the flow was so free from the wound. When this stopped and lumbar puncture was made seven days after the operation, the

leukocyte count was 26, with a trace of the globulins probably from some admixture of blood with the fluid. Culture and Wassermann both negative. This was probably a serous meningitis.

The lumbar puncture and successive leukocyte counts also give one a fair indication as to the progress of the disease, providing no intraspinal injections have been made.

Normal spinal fluid is free from the serum globulins. So if these are shown to be present by the butyric acid test, it becomes evidence of value of some meningeal process. It is always positive in purulent meningitis but, as has been shown, it may be positive in the serous form also.

When a patient with a suppurative process of the ear complains of severe headache with elevation of temperature, one ought always to suspect the presence of meningitis if careful examination fails to find other cause for these symptoms, especially if the mastoid or radical operation has been done, thereby eliminating processes which are frequently the cause of temperature and pain. A lumbar puncture should be done upon the first suspicion of meningitis as an early diagnosis is in every way desirable. For the latent types a lumbar puncture is the only method of diagnosis before the disease becomes active.

Prognosis.—The prognosis of purulent meningitis is very grave. Some patients do get well and it is hoped that some treatment may be found that will give a better outlook. At present the surgeon, while doing all in his power for these patients, hardly dares to cherish the hope that his efforts will be successful.

Treatment.—The best treatment is preventive. By an early removal of the source of infection in the mastoid, sinus or labyrinth, and by a careful operative technic which will reduce injury of the dura to the minimum, undoubtedly many patients may be prevented from developing meningitis. When it is once developed one must decide what he proposes to do in the hope of a cure,

and having formulated his plan of treatment, adhere to it. If the disease is due to suppurative labyrinthitis, the Neumann operation seems to offer the best prospect of cure, however little it may be. If the meningitis is due to other otitic causes, a subtemporal decompression offers as much hope as any surgical procedure. This may be done as in the operation for temporosphenoidal abscess (Fig. 115), only the bone exposure is more extensive over the squama. A crucial incision is made in the dura to relieve the intracranial pressure. Sutures are passed around the branches of the middle meningeal arteries on either side of the dural incision. This operation may be made part of the labyrinth operation and the dura incised later when intracranial pressure becomes increased. After the operation autogenous vaccines, proper serum therapy, or the His leukocyte extract may aid to a cure. Cases have been reported in which the patient was cured by repeated lumbar punctures, others by the intraspinal injection of urotropin and the internal administration of this remedy, and still others in which nothing was done.

Serous Meningitis.—Meningeal symptoms are sometimes present without bacterial invasion of the meninges, as, when this occurs there is generally an increase of cerebrospinal fluid, the disease is termed Serous Meningitis. Meningismus or meningeal irritation may with propriety be considered as a variety of serous meningitis, although in this condition there may be no noticeable increase in the amount of the cerebrospinal fluid.

Causes.—The causes are, suppurative processes in the temporal bone adjacent to the dura, suppurative labyrinthitis and brain abscess. An extradural abscess, the inflammation being in such close proximity to the subdural space, seems favorably located to produce an increase in the cerebrospinal fluid. A temporosphenoidal abscess, lying as it does between the surface of the brain and the ventricles is able to produce an increase in the cerebrospinal fluid in both regions. The abscess may or may not

be encapsulated, but the presence of the inflammation leads to edema of the brain substance and an exudation of serum into the ventricles or on to the surface of the brain. A cerebellar abscess may act very much in the same manner but may also lead to pressure upon the aqueduct of Sylvius or the foramina which connect the fourth ventricle with the subdural space and lead to the retention of the cerebrospinal fluid within the ventricles. Thus, dilatation of the lateral ventricle not otherwise accounted for should lead one to suspect cerebellar abscess.

The author operated upon a patient in whom this condition called his attention to the location of the abscess in the cerebellum. It was evident that some intracranial disease was present. Upon exploring the temporosphenoidal lobe, the descending horn of the lateral ventricle was opened at a depth much less than normal and ventricular fluid under increased pressure escaped. This internal serous meningitis directed attention to the probability that the disease was in the cerebellum, which was then explored and the abscess evacuated.

Pathology.—The pathological changes vary with the lesion causing the meningitis. In disease located in the temporal bone and extradural abscesses, there is a congestion of the meninges with an exudation of serum which escapes to mingle with the cerebrospinal fluid—a sort of collateral edema. In brain abscess an edema of the surrounding brain substance accounts for symptoms and cytological findings. In meningismus upon exploring the brain an edema has at times been found; at others, nothing to account for the symptoms has been discovered.

Symptoms.—The symptoms are very much the same as in the early stages of the purulent form. Pain is the predominant one, although some fever may be present. There may also be stiffness of the neck and increased reflexes with photophobia.

Diagnosis.—The diagnosis is made by lumbar puncture. If this shows a sterile fluid under pressure with no marked leukocytosis, one makes a tentative diagnosis of serous

meningitis. If the patient recovers and at no time was the culture positive, the diagnosis is justified. In meningismus or irritation of the brain, the diagnosis is made by the fact that the patient recovers from alarming symptoms, which are present at times and for which no sufficient pathological basis could be determined. It is well to state that meningismus is a name given to symptoms resembling meningitis, the cause of which is unknown, the word being more or less a "cloak for ignorance." It is hoped that it will become obsolete, as it bids fair to do.

Prognosis.—The prognosis as compared with the purulent form is good. Many patients get well. Nevertheless it may assume the purulent form, when it is usually fatal. As brain abscess may be the cause, the prognosis assumes gravity on that account.

Treatment.—The treatment of serous meningitis is first to remove, if possible, the cause. Disease in the temporal bone should be properly dealt with. A mastoid operation or the removal of an offending labyrinth may result in cure. Lumbar puncture may relieve the increased intracranial pressure or it may be necessary to accomplish this by a decompression operation. It is well to make several linear incisions in the dura instead of a crucial incision, as these may permit sufficient drainage to relieve tension and result in less brain hernia. If they are insufficient, however, the dura may be more freely incised and protrusion of the brain allowed.

Lumbar Puncture.—Lumbar puncture may be performed with the patient lying upon the side with the chest bent well forward upon the abdomen and the thighs flexed. This position opens the intervertebral spaces. The operation must be done in a thoroughly aseptic manner and with a sterile field, both on account of the danger of infecting the patient and the necessity of keeping the fluid uncontaminated for laboratory examination. The site of the puncture may be frozen with ethel chloride spray if desired, although the operation is usually done without it.

A stiff needle about three inches long is used and entered either between the third and fourth or fourth and fifth lumbar vertebræ. It is entered slightly to one side of the spinous processes and directed so that the point of the needle will pierce the intervertebral ligaments about in the median line. A sensation of resistance overcome is experienced as the needle passes through these ligaments and enters the lumbar sac, which will be at a depth of from one to two inches in adults, varying with the amount of adipose tissue. In children the depth is less.

The fluid is allowed to flow under its own pressure. No aspiration should be used nor more than 50 c.c. allowed to escape at one time without allowing an interval to elapse, as change in intracranial pressure may cause unpleasant symptoms. After-effects which at times occur are headache, weakness of the lower extremities, subnormal temperature and incontinence of urine, but as performed by the aurist upon patients with intracranial complications, none of these symptoms are at all common.

BRAIN ABSCESS.

Otitic brain abscess occurs as a result of infection and breaking down of brain substance, the primary focus being a suppurative middle-ear process.

Causation.—Brain abscess is more often associated with chronic purulent otitis media than with the acute form. Temporosphenoidal are more common than cerebellar abscesses. The condition may arise from dural inflammation matting the meninges, the infection reaching the brain directly, or it may be carried into the cerebral substance by thrombophlebitis of the veins or through the lymphatics. In these latter instances no adhesions are as a rule found in the meninges, while in those caused by direct extension the meninges are agglutinated over a greater or less area and the abscess is at times discharging into the mastoid or middle ear through the stalk formed

by these matted meninges. The opening of such an abscess may be through the tegmen tympani or antri or through the cerebellar dura. Infection may occur through the internal wall of the sinus in thrombosis of this vein, producing cerebellar abscess as a rule, although instances are reported of abscess of the cerebrum arising from this cause. A very fruitful source of cerebellar abscess is suppurative disease of the labyrinth. Probably over one-half are produced in this way, the infection extending through the internal auditory meatus, the aqueductus vestibuli or some defect on the posterior surface of the petrous pyramid.

Pathology.—Usually there is but one abscess but at times there are more. They are usually located adjacent to the infective process causing them, but may at times be at some distance, even on the opposite side in some reported cases, which are no doubt metastatic in nature. Some acute abscesses never form a capsule, while in others running a more chronic course a thick capsule may form. The capsule is composed of brain tissue infiltrated and hardened by inflammatory products and represents nature's barrier against the further extension of the process. Causes determining whether or not a capsule will be formed are the resistance of the patient and the nature of the infection. Neumann has shown that a capsule is especially apt to form in diplococcic infections, while it is a matter of common observation that they are rarely seen in infection with the streptococcus. While a capsule usually denotes chronicity, it does not always do so. Some abscesses may become encapsulated in a few weeks. In these, however, the walls of the abscess do not seem as firm as in the more chronic processes. The pus from an abscess cavity is frequently of a very foul odor. Whiting states (personal communication) that he has never observed pus in the mastoid cavity which possessed foul odor which did not come from some intracranial complication. Usually cultures may be made from the contents of an abscess cavity and colonies of bacteria grown, or

smears may show bacteria in abundance. Occasionally the bacteria are dead and will not grow upon cultivation. In such an event natural processes are no doubt making a more or less successful effort at cure. It is believed by some that it is possible for an abscess cavity to become filled with cheesy or calcarious matter and lie dormant throughout the remainder of the patient's life. Still some doubt is felt as to this being the case in instances in which it is supposed to have taken place. If, however, an abscess ruptures into the mastoid or tympanum the possibility of a spontaneous cure cannot be denied. In order that this may be possible the opening should be a large one, affording adequate drainage. Some of the cases reported, in which spontaneous cure has been believed to have taken place, are doubtless either extradural or at most subdural collections of pus, although no doubt some are true brain abscesses.

Symptoms.—Abscesses produce different symptoms during their stage of formation when there is an acute cerebritis of greater or less extent than later when a capsule may be formed and the process more or less latent. The symptoms again change in the terminal stage when the patient is dying with meningitis or edema of the brain. Moreover, a serous meningitis may complicate brain abscess and add confusing symptoms.

In the early stages pain is a prominent symptom. It is more often present than any other clinical manifestation of brain abscess. It may persist throughout the disease or until the patient becomes comatose. No deduction can be drawn from its situation as to the location of the abscess. Experience shows that temporosphenoidal abscess often occurs with occipital headache, while in cerebellar involvement the pain may be in the frontal region. It is frequently very intense and prevents the patient from sleeping. It may, however, be of a more or less remittent type or even absent or so mild that the patient does not dwell upon it.

In the early stages a rise of temperature is usually

present. The fever, however, is almost always less than in sinus thrombosis or in the majority of patients suffering from meningitis. The rise of temperature is more or less continuous at first but soon, usually in the second or third week from the beginning of the cerebral process, the temperature will drop to subnormal at some time during the twenty-four hours. It rises again to between 100° and 102° F. as a rule. This is usually regarded to indicate that the brain tissue has broken down and an abscess formed, accompanied, probably, by increased intracranial pressure. As the disease progresses more and more subnormal points will be found upon the chart. It is, however, uncommon for it to remain below normal throughout the twenty-four hours.

In the early stages the pulse is usually more rapid than normal. It is not as a rule as rapid as in meningitis. As the temperature becomes subnormal, the pulse as a rule becomes slow. It seems to vary with the temperature, although it may become slower than normal throughout the twenty-four hours. At times it is as low as 45 per minute, but a pulse around 60 in a patient suffering with a serious complication of middle-ear suppuration should suggest brain abscess. It is well to have the pulse and temperature taken every two hours, as a subnormal fall of either or both of them may direct one's attention to the true nature of the process.

Mental apathy occurs usually about the time the pulse and temperature fall, although it is not uncommon before that time. The patient seems more or less drowsy and answers questions with hesitation. He takes very little interest in what is going on around him. It is evident that his mental processes are performed with difficulty. As the disease progresses this mental apathy deepens into stupor and the patient becomes semicomatose, being aroused with difficulty.

Vomiting is a common symptom and slightly more frequently seen in cerebellar than cerebral abscesses. One readily understands this when one considers how

often a cerebellar abscess is dependent upon a manifest purulent labyrinthitis. It is a difficult matter, from a study of reported cases, to determine how often the vomiting may be due to the labyrinthine condition and how often to cerebellar abscess.

Changes in the eye-grounds usually occur in processes below the tentorium and are therefore more common in cerebellar than in cerebral abscess, although they are very often present in the latter condition. They vary from an injection of the retinal vessels to a well-marked "choked disk."

The above symptoms are common to abscess of the cerebrum and cerebellum. There are, however, certain localizing symptoms which will be discussed later. Among the other symptoms occasionally seen may be mentioned paralysis of the muscles of the eye, especially the external rectus, ptosis, change in the reflexes, various paralyses, incontinence of urine and feces, carphologia, Cheyne-Stokes respiration, etc., all marking a profound disturbance of the central nervous system. As the disease passes into the terminal stage there may be the symptoms of the purulent meningitis which not infrequently develops at the end.

Diagnosis.—In the early stage when there is fever and increased pulse rate with other symptoms pointing to intracranial involvement, it is necessary to rule out meningitis. This is done by lumbar puncture and examination of the spinal fluid. It should, however, be borne in mind that serous meningitis may be caused by brain abscess. Once having determined that in all probability the lesion is in the brain itself the question arises where it should be looked for. In the absence of the localizing symptoms it would be necessary to explore both the temporosphenoidal lobe and the cerebellum. The former would be selected if no localizing symptoms pointed elsewhere, both on account of the fact that abscesses are more common in this location, and especially because this form of brain abscess, unless on the left

side, does not give localizing symptoms, while those in the cerebellum frequently, if not usually, do so.

Localizing Symptoms.—Most of the cerebral abscesses of otitic origin occur in the temporosphenoidal lobe, although occasionally one is found in the frontal, parietal or occipital lobe. If the process is on the left side, there may be aphasia. This is usually of the amnesic variety. The patient cannot name any common article such as a key, a knife or a pencil which may be shown to him, although he knows what it is and may attempt to tell what it is used for. Once told the name he repeats it with ease. This form of aphasia is due to a subcortical lesion in the temporosphenoidal lobe and indicates with certainty the location of the abscess. If the lesion involves the cortex, there may be word-deafness as well. Occasionally, the third left frontal convolution is the seat of an abscess. This produces motor aphasia. He cannot speak the word to name the article shown him nor can he repeat it after hearing it spoken. If the lesion is on the right side, there is no aphasia or other localizing symptoms at present known unless it affects the motor area when crossed paralysis is to be expected, although this result may doubtless be produced by pressure upon the internal capsule.

Cerebellar Abscess.—In cerebellar abscess the patient is very apt to have ataxia and may also have nystagmus. When standing on one foot or on both feet with eyes closed, or in hopping forward and backward, the patient is supposed to have a tendency to fall to the involved side, and as a matter of fact usually does so.

In nystagmus of cerebellar origin, the nystagmus is usually toward the involved side. A great deal has been written about this nystagmus and in what part of the cerebellum the lesion must be to cause it. Wilson[1] regards it as ataxic in nature and unlike labyrinthine nystagmus; it is less behind opaque glasses. If a patient with cere-

[1] Trans. Amer. Otol. Soc., 1915.

bellar abscess has nystagmus to the same side, which is the rule, he falls in the direction of the rapid component of his nystagmus. In nystagmus of vestibular origin the patient falls in the direction of the slow component and also in the direction which the slow component assumes if the position of his head is changed. Suppose the patient has spontaneous rotatory nystagmus to the left of vestibular origin or that it has been produced by syringing the right ear with cold water. As the nystagmus is to the left, therefore the quick component is to the left, the slow component is toward the right if the face is directed forward and the patient falls to the right. If the face is directed over the left shoulder, the slow component is forward and the patient falls forward; if the face is directed over the right shoulder, the slow component is backward and the patient falls backward. In cerebellar abscess whether or not nystagmus is present and no matter what its direction if present, the patient falls to the involved side and the position of the head does not change the direction of falling.

Another symptom which may be present in cerebellar disease is overpointing. When present, if the patient places the forefinger of the hand corresponding to the side of the lesion on the finger of the examiner and is then requested to raise or lower the finger and bring it back to the finger of the examiner he will do so quite accurately as long as he guides it by the sense of sight. When he closes his eyes, his finger will not return accurately to its original position, as in the normal person, but will deviate outward, that is, toward the side corresponding to the lesion. This may be tried with a wrist motion by holding the forearm, with a movement at the elbow-joint by holding the arm or by motion at the shoulder-joint. Great reliance can be placed on this overpointing, as when present it indicates disease of the cerebellum. In horizontal nystagmus of vestibular origin the patient overpoints in the direction of the slow component.

If nystagmus is present and directed toward the sup-

posed side of the lesion, and if the labyrinth on this side is inactive, as shown by the tests, and if at the same time there is slow pulse and subnormal temperature, it is the surgeon's duty to explore the cerebellum. This clinical picture would be present in cerebellar abscess following a suppurative labyrinthitis.

Another test for ataxia is known as adiadokokinesis. The patient is requested to alternately pronate and supinate both hands as rapidly as possible. This he can do only in a very deliberate manner on the side of the lesion if the test is positive through cerebellar ataxia, while on the opposite or normal side he will be able to do it quite rapidly.

Another symptom of cerebellar abscess which occurs at times is paralysis of respiration. The breathing ceases but the heart continues to beat and will do so for hours if artificial respiration is maintained. In numerous instances this has occurred at the beginning of an operation and has led the surgeon to open the cerebellum and evacuate an abscess after which the respiratory movements took place normally again.

Prognosis.—The prognosis of brain abscess is grave. More than one-half of the patients die. Abscesses giving the best prognosis are those with a stalk with pus already discharging into the mastoid or middle ear, and those in which the meninges are matted together, thus lessening the danger of meningitis after operation.

Treatment.—The treatment is the surgical evacuation of the abscess and maintenance of drainage until the abscess cavity heals.

Operation, Cerebral Abscess.—The approach to a temporosphenoidal abscess may be internal, through the roof of the mastoid and tegmen tympani, or external through the squama. The statistics of operation by the internal route are more favorable on account of the fact that this is used to drain abscesses with stalks.

Internal Approach.—The internal operation is performed as follows: The dura is exposed by removing the

inner table over the tegmen of the mastoid and tympanum, a radical operation having been previously performed. If a fistula is found discharging, it is enlarged, taking care not to invade the meninges beyond the point of agglutinizing adhesions, as infection may cause meningitis. A cigarette drain or folded rubber tissue may be inserted. If no stalk is found, an incision is made in the dura and a brain knife or grooved director inserted into the brain substance. The point of entrance of the instrument should be on the summit of a convolution and not in a sulcus. If the latter situation is selected, the pial vessels which are abundant in the sulci will be injured and the hemorrhage will obscure the field and render it uncertain whether or not a small amount of pus has escaped. For the same reason any vessels in the dura are to be avoided when making the incisions. If the knife is used, after it has entered the brain to a sufficient depth, usually about an inch, although in some directions it is safe to go farther, it is rotated so that the surfaces of the incision are held apart and the pus if present allowed to escape. If pus does not come from one puncture, several may be made in different directions. As long as the knife or grooved director is inserted directly into the brain and brought out in the same line, no harm results, but if the instrument is moved laterally while in position the brain tissue will be broken down and harm result. If the pus flows, the opening is enlarged and a drain inserted. Nothing of value will be accomplished, as a rule, by inserting the finger or encephaloscope. Once the abscess is opened and drainage is established the surgeon's duty is simply to see that it is properly maintained.

External Approach.—In exploring the brain by the external route an incision is made from the mastoid incision upward over the squama and the flaps retracted in either direction after being elevated from the squama. An opening is made with the large gouge and enlarged with the rongeur. A small dural incision is made, as a large one will result in the production of a larger brain

hernia during the healing process. The brain is explored as in the internal route. Fig. 115 shows the bone removed as in the two operations. The lower part over the mastoid and middle ear for the internal operation, while the upper part in the squama for the external operation.

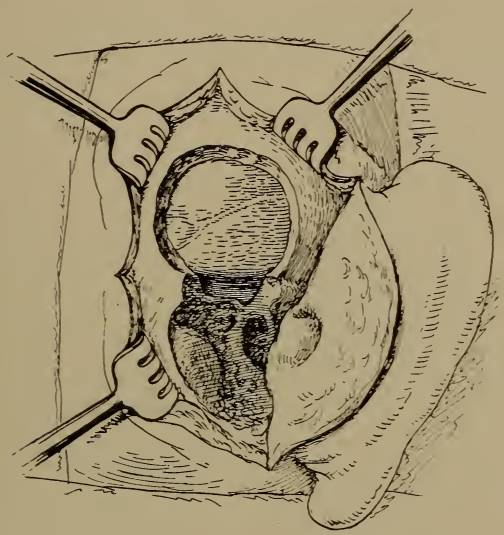

FIG. 115.—Operation for temporosphenoidal abscess; also subtemporal decompression. The shaded portion of the exposed dura is part of the floor of the middle fossa.

Combined Operation.—It is frequently a good plan to make the exploration at the junction of these two on the external and inferior border of the temporosphenoidal lobe. This is much more convenient and many successful operations have been done in this manner. One is then able to direct the exploring instrument in any direction desired.

If the abscess is located in the motor area for speech, the bone removal may be continued forward and the abscess evacuated, or an incision may be made directly over this area, the bone removed and the brain explored.

The same may be done if the abscess is in the cortical center for the face, arm or leg muscles. But these abscesses are very rare as complications of middle-ear suppuration.

Operation for Cerebellar Abscess.—In the cerebellar operations there are also two routes; one anterior and internal to the sinus and the other posterior to the sinus through the occipital bone. If the abscess has followed sinus thrombosis, it may very conveniently be opened through the inner wall of the sinus. The anterior operation may be very difficult if the sinus is far forward, as

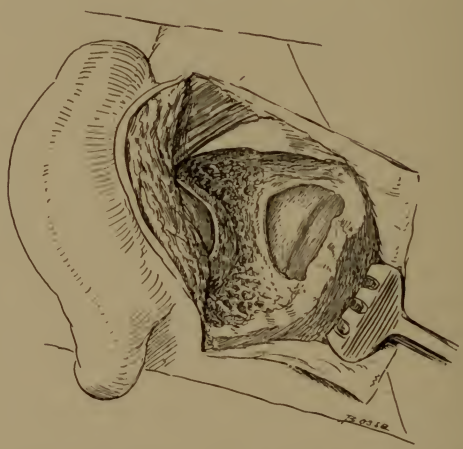

Fig. 116.—Trautman's triangle. The shaded portion is the lateral sinus.

one then has very little room in which to work. The statistics of cerebellar abscess show that the mortality is less in drainage by this approach, mainly because abscesses with stalks are found discharging into the mastoid through this route.

Antero-internal Approach.—The first step is to expose the dura in Trautman's triangle (Fig. 116). The base of this triangle is the internal anterior border of the lateral sinus which is partially shown as the shaded portion of the dura in Fig. 116, the upper border is the inner table or dura of the middle cranial fossa, its lower

border being the lower limits of the mastoid, while its apex is inward at the posterior semicircular canal. If there is a stalk to the abscess, it is treated in the same manner as in the internal operation for temporosphenoidal abscess. If not, the cerebellum is explored through a small dural incision. The knife may be passed inward along the posterior border of the petrous pyramid, and in other directions if pus is not evacuated from this location.

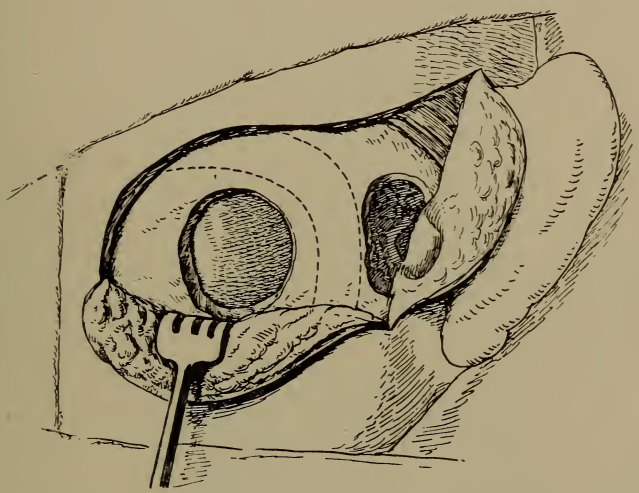

FIG. 117.—Posterior operation for cerebellar abscess. Dotted lines show location of sinus.

Posterior Approach.—If the sinus is well forward or as a matter of election, the posterior route may be used. A large flap of skin and muscle is turned down, making the incision from the mastoid wound backward toward the external occipital protuberance, but before reaching this landmark turning downward. This large flap is separated from the bone and an opening made, thus exposing the dura over an area from one to two inches in diameter (Fig. 117). This should reach forward nearly to the lateral sinus. Most aural surgeons begin this with the

gouge and complete it with the rongeur, but a trephine may be used if desired, although the bone is usually not very thick in this region. A small dural incision is made and the cerebellum explored. It is necessary to bear in mind, while exploring backward and downward, that this field is much nearer the fourth ventricle than when operating anterior to the sinus, and the instrument should not be passed over an inch in this direction.

After the abscess heals, if the patient recovers, the flap may be returned to its position by a plastic operation and be very convenient in the treatment of brain hernia, as moving a flap of skin and subcutaneous tissue is often a successful way to treat this complication of the healing of brain wounds.

After-treatment.—In the after-treatment the drain may be shortened from day to day as the abscess cavity fills up. Retention of the pus should be guarded against. At times a patient fails to recover on account of a second abscess which was not drained at the original operation. These are to be sought and opened if possible.

FACIAL PARALYSIS.

Facial paralysis of otitic origin may occur during the course of a suppurative middle-ear process, or as the result of injury during operative procedures, or may first appear during the healing process subsequent to such intervention.

Pathology.—The facial nerve occupies a closed canal from the internal auditory meatus to its emergence at the stylomastoid foramen. Throughout the greater part of this canal it is in intimate relation with the tympanum and mastoid. Suppurative processes in these parts not infrequently extend to the nerve, producing a neuritis with loss of function. At times there is a dehiscence in the Fallopian canal in its tympanic part, through which an infective process readily gains access to the nerve.

Symptoms.—In a gradually developing facial paralysis there first appears a weakening of the muscles of the face.

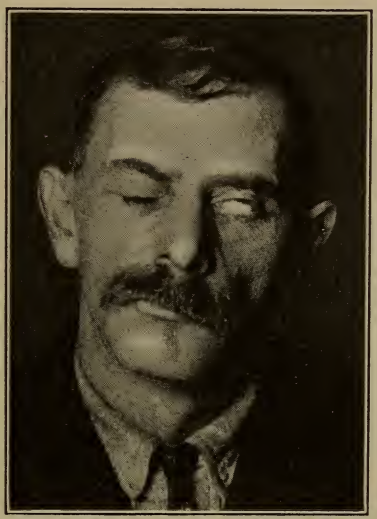

FIG. 118.—Facial paralysis. Attempting to close the eyes. (Jelliffe.)

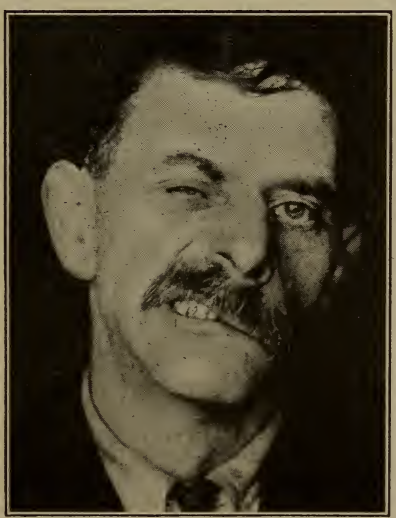

FIG. 119.—Facial paralysis. Attempting to show the teeth. (Jelliffe.)

The patient cannot close the eye tightly and with the same force as upon the opposite side, or the movements of the muscles about the mouth on the involved side are not so free. He cannot expose his teeth as much on this side as on the other. He may still be able to whistle but does so with difficulty. As the paralysis deepens and finally becomes complete, he will be unable to close his eye (Fig. 118) but in the attempt to do so rolls the cornea beneath the upper lid. He loses the ability to move the angle of the mouth and consequently cannot show his teeth (Fig. 119). Whistling becomes impossible and food may collect between the cheeks and the teeth, producing difficulty during mastication.

Chorda Tympani.—If the lesion causing the facial paralysis is in the Fallopian canal, the chorda tympani is involved also. This causes loss of taste sense on the anterior two-thirds of the tongue. While there is considerable variation in the acuity of perception of the various sapid substances on the different parts of the gustatory surface, still in testing taste perception a solution of sufficient strength is used to enable the patient, if normal, to recognize the taste if applied to any part of this surface. If applied to the anterior two-thirds of the tongue, a strong solution of quinine sulphate is recognized as bitter, a 50 per cent. solution of sodium chloride produces the sensation of salt, simple syrup appears sweet, and a 25 per cent. solution of tartaric acid is recognized as sour. In facial paralysis, if the lesion is located in the geniculate ganglion or between it and the stylomastoid foramen, or even contiguous to this foramen, the patient becomes unable to appreciate these primary tastes.

Manner of Making Tests.—It is found that if a patient is requested to protrude his tongue and one of the solutions is applied, with a cotton-tipped applicator, to its anterior two-thirds and he is then asked to state the sensation, in attempting to speak he will withdraw his tongue, spreading the solution over other parts of the

gustatory surface. The experiment thus becomes of no value. To obviate this a chart is printed with the words, sour, salty, bitter, sweet, and after the solution is applied the patient is requested to point to the experienced sensation without withdrawing his tongue. One thus ascertains whether or not he recognizes the taste of the solution applied to the anterior two-thirds of his tongue and thus forms an opinion as to the integrity of the chorda tympani.

Diagnosis.—The diagnosis of facial paralysis offers no difficulties. One is able to see at a glance that the muscles of the face do not contract normally. It is important to determine the location of the lesion if possible, not only as bearing upon the prognosis of the paralysis but also as having an influence upon the treatment adopted for the process of which it is a complication. The occurrence of facial paralysis may determine the adoption of operative interference in a given case. There is, however, no reason why a patient with a suppurative middle-ear process may not have facial paralysis from involvement of the nerve in the neck. So before relying upon facial paralysis as a symptom which indicates operation, a test of taste perception of the anterior two-thirds of the tongue should be made. The writer has observed two patients with suppurative middle-ear disease and facial paralysis, upon whom the mastoid operation would have been done, but for the fact that the taste sense was not lost on the anterior two-thirds of the tongue. The chorda tympani thus being intact, the facial paralysis was not due to the suppurative process, the mastoid was not opened and recovery without operation took place. If the tests show involvement of the chorda tympani, the facial paralysis is not necessarily due to the middle-ear process, but no means are known to determine that this is not the case. Tests show that there is loss of taste in about 10 per cent. of acute and nearly 50 per cent. of chronic middle-ear suppurations independent of facial paralysis.

Prognosis.—Those paralyses which develop before operation usually offer a good prognosis. Occasionally one develops with labyrinthine involvement and is due to sequestration of a portion of the Fallopian canal. In this case the outlook as to the cure of the paralysis is not good. Paralyses which are present at the conclusion of an operation are usually due to some injury of the nerve and do not offer a good prognosis. The palsy may exist at the end of the operation and may not be due to injury inflicted by the instruments of the surgeon, and at times a patient with this condition recovers the action of the facial muscles to a large extent. Nevertheless, when facial paralysis is present at the end of an operation, more or less permanent impairment of function may be expected. Those paralyses which develop subsequent to operation offer a good prognosis, unless due to sequestration of the labyrinth.

Treatment.—If the facial paralysis appears before operative interference and is due to the purulent otitis media or mastoiditis, it is usually an indication for an operation. Taken alone without any indication that the process which it accompanies is serious, it does not demand operation either for the cure of the paralysis or of the process of which it is a complication. Still, one is so accustomed to see facial palsy only in processes of a serious nature that he is usually uneasy until operation is performed. The paralysis, if not due to injury of the nerve, usually begins to get well in about six weeks. Meanwhile, the nutrition of the muscles may be maintained by the daily use of electricity and massage. This is especially necessary when recovery does not take place so promptly.

Indications for Anastomosis.—When due to injury the advisability of forming an anastomosis between the facial and the hypoglossal or spinal accessory is to be considered. It is difficult to say just how soon after the occurrence of the injury this should be done. In a patient at the New York Eye and Ear Infirmary, whose

paralysis was evident on his emergence from the anesthetic, total facial paralysis persisted for nearly two years, when improvement began and continued until the facial muscles acted in almost a normal manner. The final condition of this patient was perhaps much better than it would have been had an anastomosis been made. Experience has shown that when one waits too long after the paralysis has occurred, the result is not so good as in an earlier operation. After the reaction of degeneration is present for a certain length of time, the nerve seems to become softer and is not so easily handled. If the operator is certain that the nerve is completely divided, he is not justified in waiting so long as when he believes that only partial division has taken place. In the latter case it is probable that the total paralysis is due to an inflammation of the uninjured part of the nerve, and partial, or at times practically complete, return of function may be expected.

Anastomosis.—The first anastomoses were made between the spinal accessory and the facial, either an end-to-end union or implantation of a part of the spinal accessory into the facial. In the former case paralysis of the sternomastoid and part of the trapezius takes place; in the latter after healing has occurred there may be involuntary movements of the shoulder when the patient attempts to move the face. For this reason, implantation of the facial into the hypoglossal has become the operation usually selected. An incision is made along the anterior border of the mastoid immediately behind the auricle and extending downward along the anterior border of the sternomastoid which is exposed and drawn backward. The posterior belly of the digastric is then located. The styloid process may be felt with the finger. The nerve emerges from the bone at the base of this process and passes superficial to it. It is well to remove the bone over the descending limb of the Fallopian canal and carefully remove the nerve after cutting it as high as possible. This gives a longer stump and allows the

connection to be made with the hypoglossal without tension. The hypoglossal is now sought, the guide being the transverse process of the atlas. The occipital artery may be felt, and if traced downward to its origin from the carotid, the nerve may be located winding around the artery, but the anastomosis is made above this point. The jugular vein also comes into view and is to be drawn backward, when two nerve cords are exposed, the pneumogastric and the hypoglossal. By electrical stimulation the hypoglossal is selected. This is recognized by the contraction of the muscles of the tongue and thyroid group. A slit is made in the hypoglossal and into this slit the stump of the facial, previously trimmed to a point, is inserted as follows: two stitches are taken in the facial stump a short distance from the end, one on either side, these sutures are caught in the margins of the slit in the hypoglossal and tied as the facial stump is inserted into the slit. Cargyle membrane is placed around the union to prevent scar formation, and the wound closed. The hypoglossal paralysis passes away more or less completely. The patient is submitted to various educational exercises to accustom him to the use of the new nerve path.

ABDUCENS PARALYSIS.

Paralysis of the external rectus is at times associated with and due to suppurative middle-ear disease. The author[1] collected all of the cases reported in the literature of the subject, a total of 88, to which he added 6 cases, making a total of 94. Since that time numerous cases have been reported by various observers.

Anatomy.—The anatomical arrangement of the sixth nerve renders it susceptible to involvement in the purulent diseases affecting the temporal bone. From a point where the nerve enters a perforation in the dura, external to the inferior petrosal sinus, until it comes in relation to

[1] Annals of Otology, etc., September, 1910.

the branches of the third nerve on the external wall of the cavernous sinus, it is contained in a fibrous canal called by Gradenigo, Dorello's canal, from the anatomist who first described it. As it passes forward it is in relation with the inferior petrosal sinus which lies immediately internal to it, then it passes beneath a bridge which is shown cut in Fig. 120. This bridge is formed

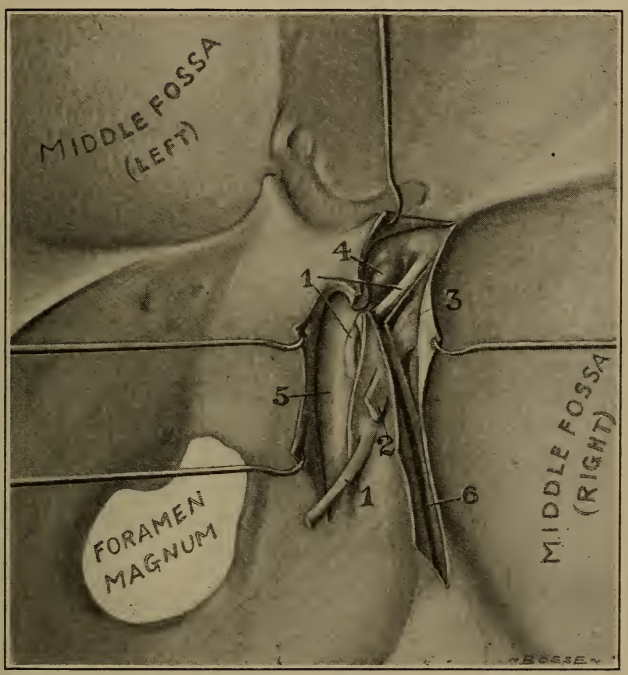

FIG. 120.—Relations of the sixth nerve. *1*, sixth nerve; *2*, fifth nerve; *3*, Gasserian ganglion on fifth nerve; *4*, internal carotid artery; *5*, inferior petrosal sinus; *6*, superior petrosal sinus. The petrosphenoidal ligament is shown cut in the figure.

by the petrosphenoidal ligament which extends from the internal end of the superoposterior border of the petrous pyramid to the base of the posterior clinoid process. Beneath this bridge and slightly anterior to it the nerve lies upon the extreme end of the apex of the petrous portion of the temporal bone. Immediately after passing

the bridge it is in relation above with the superior petrosal sinus and externally with Meckel's cave which contains the Gasserian ganglion. From this point forward it lies on the outer wall of the cavernous sinus, emerging at its anterior end to enter the orbit through the sphenoidal fissure.

Pathology.—It is evident that thickening of the fibrous tissue which encloses the nerve will result in pressure upon, and interference with, the function of the nerve. This may take place through inflammatory processes in the posterior fossa. In this event the swelling may be of the dura at its point of entrance, as in large extradural abscesses in the posterior fossa, or from thickening of the tissue around the nerve, as in thrombophlebitis of the inferior petrosal sinus. Where it lies upon the apex of the petrous portion any inflammation of the bone which thickens the tissue surrounding the nerve leads to abducens paralysis. In some pneumatic bones the cells extend well into the petrous portion, and there is not infrequently a large cell immediately at the tip adjacent to the nerve (Fig. 53). With such a conformation the involvement of the mastoid cells and middle ear may extend sufficiently to involve this cell also. In doing so the infection may take one of the following paths: (*a*) sublabyrinthine, extending below the labyrinth and internal auditory meatus, as in a patient dying of meningitis, whose case was reported by Lombard.[1] Autopsy showed very pneumatic bone with pus in the cells of the petrous apex. On the opposite side the cells in the apex of the petrous portion were large and mercury poured into them emerged in the tympanum; (*b*) from the mastoid antrum the infection may extend through the subarcuate fossa and by involving the chain of cells that sometimes exist above the internal auditory meatus, thus reach the cells in the petrous apex; (*c*) along the carotid canal; (*d*) or by way of a layer of cells extending along the

[1] Annals des Mal. de l'Oreill, etc., 1906, xxxii, 321.

Eustachian tube. After pus has formed in the petrous apex it may break through superiorly, forming an extradural abscess, or inferiorly escaping posterior to the nasopharynx, forming a retropharyngeal abscess.

Symptoms.—There are symptoms of the middle-ear process of which the abducens paralysis is but an incident. The affection of this muscle is easily made out. As the eyes fixed upon the finger follow it outward, the one involved does not abduct, thus showing paralysis of the external rectus supplied by the sixth nerve. If the affection is long-continued convergent squint may exist. As the Gasserian ganglion lies adjacent to the cells in the petrous apex, it may be simultaneously involved with the sixth nerve when the lesion producing the paralysis occurs in this location, causing pain in the parts supplied by the fifth. This is very marked in some patients and the association of severe neuralgic pain referred to the region of distribution of the fifth nerve, with abducens paralysis and suppurative otitis media, is called Gradenigo's triad, from the otologist who first described these associated symptoms and recognized their significance.

Diagnosis.—The diagnosis offers no difficulties. If the paralysis of the external rectus has occurred with, or following upon, a suppuration of the middle ear, it is reasonable to suppose that it is in some way related to it, unless another cause can be discovered. If in addition there are severe neuralgic pains of the brow or face, the lesion is in all probability located in the apex of the petrous portion.

Prognosis.—Of the 94 patients, 11 died of meningitis, recovery occurred in 67, probable recovery in 5, partial in 4, no improvement in 1, not stated, 6.

Treatment.—Involvement of the sixth occurred after the mastoid operation in 27 patients, not stated, 7, and before in 60. Of these 60 the operation was performed in 29 with 1 death, 1 persistence of the paralysis, while in the remaining 27 the condition of the abducens became

normal. If a mastoid operation has not previously been done, it is no doubt indicated if the patient has acute otitis media. If suppuration is chronic, the radical operation should be performed. Still it must be remembered that patients do recover without operative interference. The operation performed by Goris[1] demonstrates that the region of the petrous apex is not beyond surgical interference. In this procedure the roof of the external auditory canal, tympanum and mastoid are removed, thus exposing the dura to the superior semicircular canal. From this point in, the dura is separated from the bone and held away by spatulæ, the petrous tip curetted, or an extradural abscess evacuated. Goris, in the patient upon whom he operated, found an extradural abscess and with a curette removed a sequestrum from the petrous apex. Abscesses in the pharynx or neck should be evacuated either by incision through the mouth or by an external route, according to their location or preference of the surgeon. Incisions seem to be, as a rule, all these abscesses require.

[1] Annal. des Mal. de l'Oreille, etc., xxix, 64.

CHAPTER XII.

NON-SUPPURATIVE DISEASES OF THE INNER EAR.

General Considerations.—Diseases of the labyrinth and eighth nerve, notwithstanding much important work, still remain more or less obscure. While the lesions which complicate the suppurative inflammations of the middle ear and mastoid are becoming better understood, progress is made slowly in the knowledge of those diseases of the inner ear which do not occur with these processes. One reason is that these diseases are perhaps not so common, but more important, still, the fact that opportunity of examining the temporal bone of patients carefully observed during life rarely occurs.

Causation.—When the perceptive mechanism is involved the deafness is usually quite marked. Many of these patients give no history of attacks of any kind to which the deafness may be attributed. They may or may not give a family history of deafness. Many of them are doubtless subjects of otosclerosis. The Wassermann should always be taken and throws light on the causation at times. Some patients give a history of having impairment of hearing or total deafness following some general disease. Many of these seem to be able to produce sound-perception mechanism disease without apparently involving the middle ear. Epidemic cerebrospinal meningitis, scarlatina, measles, diphtheria, mumps, smallpox, typhoid fever, and others, are capable of causing such processes.

The eighth nerve and labyrinth are affected by many drugs. Quinine, salicylic acid, arsenic, lead, alcohol and tobacco, are able to produce some change in these structures which cause symptoms. Usually the change is but

temporary and passes away, but this is not always the case. One sees from time to time patients who attribute their deafness to quinine or salicylic acid. Usually one is able to prove that this belief is without foundation by finding that the lesion is in the sound-conducting mechanism, but occasionally it is found to be in the receptive mechanism when one feels that there may be some basis for their belief.

Symptoms.—The symptoms may be divided into two classes: Those which arise from disease of the perceptive mechanism, namely, impairment of hearing and tinnitus, and those which arise from disturbance of the static mechanism, namely, nystagmus, vertigo, nausea and vomiting and unsteady gait. These two classes of symptoms may occur together in the same patient or impairment of hearing may be present, the patient never having had symptoms referable to the static labyrinth or vestibular nerve, in which event the static apparatus may give the normal reactions, or as is usually the case, they may be negative. The absence of caloric and a negative rotation reaction indicate disease of the static labyrinth or the vestibular branch of the eighth nerve. In order that the nystagmus, vertigo, etc., may be produced, it is believed to be necessary that the lesion producing them should develop in a more or less rapid manner. The number of patients in whom the caloric and rotation tests are negative and who give no history of having had vertigo, is considerable. They encourage the belief that if the loss of the static function is sufficiently gradual, the opposite labyrinth may be able to accustom itself to this loss without at any time manifesting symptoms of disharmony. If both sides are involved equally and make the same progression toward loss of function, it is apparent that no disharmony of the two static mechanisms will occur, but that other parts of the nervous system will, so far as they are able, take upon themselves the functions which have been lost.

In diseases of the cochlea which have produced deafness,

the bone conduction with a 256 D. V. fork should be about one-half of air conduction. If this should be the case in a patient under forty-five years of age, who had marked impairment of hearing, the lesion could with certainty be located in the perceiving mechanism. As the air conduction would be very much reduced on account of the patient's deafness, it follows that the bone conduction, which is $\frac{1}{2}$ A. C., would be reduced also (B. C.−). In diseases of the cochlear branch of the eighth, it is believed that the ratio of air and bone conduction becomes still further changed, so that B. C. becomes less than one-half of A. C. It is certain that it does in syphilitic affections of the nerve and it is probable that it does in other conditions also, although too much reliance cannot at present be placed upon it in attempting to separate lesions in the cochlea from those in the nerve.

If in marked deafness the lower-tone limit is normal, it merely is evidence that the lesion is in the sound-perceiving mechanism, but if at the same time the upper-tone limit is reduced, it indicates that the lesion involves the labyrinth, although the nerve central to the cochlea may at the same time be involved. The cochlea contains the mechanism by which the tones of the musical scale are analyzed, so if the perceptive apparatus fails to pick up and register tones of a certain pitch while others are heard, the lesion must be in the cochlea. Thus if the upper tones cannot be perceived, or if there is a gap in the musical scale in which the sonorous vibrations are not registered, or if deafness for a greater part of the musical scale exists, while tones of a certain pitch in one or more parts of the musical scale can be heard, the lesion must be in the labyrinth. If one cannot find these tone islands, tone gaps or lowering of the upper tone limit, he is not justified in assuming from this fact alone that the lesion is not in the cochlea, as instances are common where disease affects all parts equally of the organ which picks up and registers the notes of the musical scale, that is, the organ of Corti.

The upper-tone limit is lowered in those processes in the labyrinth which are secondary to non-suppurative disease in the middle ear, although it is probable that many of these which are considered to be due to chronic catarrhal otitis media are really cases of otosclerosis.

Tinnitus in some instances is a prominent symptom. If the tinnitus is due to a stimulation of the acoustic nerve or apparatus, one can readily understand why it would be present while the lesion is being formed and the deafness is increasing, but one would expect when the patient became absolutely deaf it would cease. As a matter of fact it often does so, but instances are by no means rare in which the subjective noises have persisted long after deafness has become absolute.

If every patient presenting with impairment of hearing, with or without tinnitus, and in whom no suppurative process is found, were to have a careful examination to determine the bone conduction and tone limits, it would be discovered that many patients are being treated as chronic catarrhal otitis media who have some lesion of the sound-perceiving mechanism. The diagnosis of O. M. C. C. should never be made without this examination. It is, however, much neglected in many of the large clinics. Unless the patient complains of vertigo or some symptom which directs attention to the labyrinth, the impairment of hearing is carelessly attributed to disease of the conducting mechanism and the patient treated for chronic catarrhal otitis media. In private practice this is naturally not so apt to occur.

Ménière's Symptom-complex.—In 1861 Ménière reported a case of labyrinthine hemorrhage; the patient, a girl aged sixteen years, who "caught cold" while driving and suffered an attack, the symptoms being tinnitus, deafness, vertigo, vomiting and unsteady gait. Death occurred five days later and the postmortem revealed a red plastic exudate in the semicircular canals extending into the vestibule. These symptoms having such a definite pathological basis naturally attracted attention at a time when

knowledge of diseases of the labyrinth was meagre, and for many years a patient presenting a sudden attack of deafness, vertigo, vomiting and staggering gait was supposed to have labyrinthine apoplexy, or "Ménière's disease." As it became known that the same symptoms occurred without labyrinthine hemorrhage, may in fact be due to many different lesions of the labyrinth or eighth nerve, these clinical manifestations came to be called Ménière's symptom-complex. Any lesion which produces an imbalance of the static mechanism, either by stimulating one side, or what amounts to the same thing, by cutting out the impulses which normally emanate from the other static labyrinth, will result in the production of this symptom-complex, the deafness being caused by the cochlear involvement. The feeling is quite general among otologists that the use of the term is not conducive to scientific accuracy. One very important symptom which was omitted from the descriptions of the symptom-complex is nystagmus. This is probably never absent and if the term is continued in use it must be added. It is better perhaps not to use the term at all, but to apply Ménière's name, if it must be used, to labyrinthine hemorrhage.

LABYRINTHINE HEMORRHAGE (MÉNIÈRE'S DISEASE).

Red blood cells or even small hemorrhages may be present in the labyrinth in the severe form of serous or in purulent labyrinthitis, but they constitute but a small part of the process and in themselves do not noticeably affect the symptomatology. It is to those hemorrhages which occur without infection and are the cause of symptoms that the name is applied.

Etiology.—Small hemorrhages may occur in the general infectious diseases or in the acute middle-ear inflammations, but as a rule some change in the bloodvessels or in the blood itself acts as a cause. Thus various con-

stitutional diseases predispose the patient to local hemorrhages. Insofar as the labyrinth is concerned, leukemia seems to be the chief of these. Perhaps the reason this is apparently so is that autopsies are more frequent in patients with this disease than in those who have labyrinthine hemorrhage as a result of some less fatal malady. Alexander[1] claims that Ménière's original case was leukemic. Gruber[2] reports the findings in an autopsy in a syphilitic patient who died of typhus in whom labyrinthine hemorrhage was present.

Pathology.—The lesion is the rupture of a bloodvessel supplying the membranous labyrinth, which gives rise to an extravasation of greater or less extent. It is rational to suppose that the size and location of the clot, as well as the amount of destruction or displacement of the delicate intralabyrinthine structures, will have an influence not only upon the symptoms but also on the healing process. Hemorrhage would necessarily be limited by rise of labyrinthine pressure caused by it. As the extravasation is taking place, various distortions may occur. Thus, if the hemorrhage is into the cavity of the membranous labyrinth it can make very little provision for increased pressure. It has been seen that the mechanism for regulating the pressure of the endolymph is the ductus endolymphaticus, which is a bulb containing endolymph which projects into the cranial cavity through the aqueductus vestibuli. This would be inadequate to make provision for a hemorrhage of any considerable (relatively) size so there would be distortion at other locations where this is possible. This distention in the cochlear duct results in disturbance of the delicate structures contained therein, which are concerned with hearing, perhaps also rupture of Reisner's membrane which is the most vulnerable part of this duct. This would account for the deafness in Ménière's patient in whom the exudate was

[1] Zeitschrift für Ohrenheilk., 1906.
[2] Lehrbuch der Ohrenheilk., 2 Aufl., 1881.

limited to the semicircular canals and vestibule. As the extravasation is absorbed very slight changes may be left, or adhesions between and distortions of the various intralabyrinthine structures with the formation of varying amounts of fibrous or even osseous tissue may result.

Symptoms.—The symptoms are tinnitus, deafness, vertigo, nausea and vomiting, staggering gait and nystagmus. Usually the hearing is normal before the first attack. This is insisted upon by many authorities. There seems no good reason, however, why an attack of labyrinthine hemorrhage should not occur in a patient the subject of a catarrhal middle-ear process. After the first attack more or less deafness may be expected. All of these symptoms may not be present in every patient and there is considerable variation in the severity also. After the attack the symptoms may disappear in a few days, leaving the patient totally deaf. This occurs if the hemorrhage is sufficient to destroy the intralabyrinthine structures. In such a patient the caloric and rotation tests are negative. It is more common for the symptoms to disappear gradually, the patient having nystagmus and more or less vertigo for some time. Or these may continue in a varying degree of severity until subsequent attacks occur. The condition of some of these patients becomes exceedingly distressful. If they turn around quickly or stoop over, vertigo ensues and the uncertainty as to the occurrence of the next serious attack renders life all but unbearable.

Diagnosis.—There is no certain way of ascertaining that the symptoms are due to a labyrinthine hemorrhage. One infers that the lesion is in the labyrinth from the absence of involvement of the facial which would be expected to occur if the lesion was in the internal auditory meatus, and from the absence of symptoms referable to implication of the nerves in relation with the eighth at the base of the brain. The sudden onset of the symptoms in an apparently normal middle ear is also a point in favor of apoplexy of the labyrinth.

Prognosis.—The patient may have but one attack. This is more apt to be the case if the labyrinthine functions are ablated. If recovery with some function occurs, more attacks may be expected, although they may not occur.

Treatment.—During the attack the patient is put to bed. The head, to which cold applications are made, is slightly elevated. A laxative may be administered and full doses of the bromides given. As the attack passes off the iodides may be associated with the bromides. Pilocarpine will be found of value in some patients. It is administered as suggested under Perilabyrinthitis. If the vertigo between the attacks is severe and there seems no prospect of relief otherwise, the labyrinth may be destroyed. It must be explained to the patient that he will be totally deaf but that the prospect of relief from his vertigo is good, although not absolutely certain. The decision is to be left with him. The operation is to be performed about the same as in drainage of the labyrinth. The object is to destroy the cristæ ampullæ, so the cochlea need not be molested, but the canal system and especially the ampulla of the canals must be opened and the nerve tissue as far as possible destroyed.

LEUKEMIC DISEASE OF THE INNER EAR.

Leukemic involvement of the labyrinth, while not so common as that of the retina, takes place in about 10 per cent. of the patients. The lesion in the labyrinth may be a hemorrhage or the exudation of lymph cells; as these become absorbed, new connective tissue may form or an osseous deposit occur, more or less obliterating the labyrinthine spaces. Exudation of lymph cells or hemorrhage may occur in the internal auditory meatus affecting the eighth nerve, perhaps also the seventh, or it may be in the middle ear or Eustachian tube.

Leukemic affections of the labyrinth and eighth nerve either begin suddenly with deafness, nystagmus and vertigo or the impairment of hearing may occur alone

and get worse rapidly, leading to total deafness in the course of a few weeks or months. If the lesion is in the middle ear and the sound-perceiving mechanism is intact, there may be pain, but the deafness is not so profound, nor will there be present the nystagmus or vertigo. The prognosis both as to the aural condition and the general disease is bad. If the middle ear is involved and tension is marked, an incision of the drum membrane is indicated. The treatment otherwise is the same as in labyrinthine hemorrhage, except that an operation is not to be considered.

ANEMIA OF THE LABYRINTH.

Anemia of the labyrinth is usually a part of a general anemia but it may be of a more or less local nature.

Causation.—Symptoms referable to it are usually caused by the sudden loss of a large quantity of blood, pernicious anemia, or after severe acute diseases, less often in the anemias of chronic disease. Processes causing local anemia are those which produce any interference with the flow of blood through the internal auditory artery, which constitutes practically the only blood supply of the labyrinth. Conditions which have been known to produce this result are: aneurysm of the basilar artery, neoplasms growing from the dura in the internal auditory meatus or in the immediate neighborhood, bony outgrowths in this canal, or thickening of the vessel in arteriosclerotic processes. It is probable that the latter condition is often a causative factor in the deafness of old age. An embolism, if sterile, might cause anemia, especially if small enough to reach some of the branches of the internal auditory artery so that some collateral circulation could take place within the labyrinth. Angioneurotic processes may be so localized as to effect the labyrinthine circulation.

Symptoms.—If the onset of the anemic condition in the labyrinth is acute, there will be sudden deafness which is more or less pronounced, perhaps tinnitus, vertigo and

vomiting, accompanied, of course, by the symptoms of the disease causing the anemia. The symptoms very much resemble seasickness. In the angioneurotic form, which, presumably, would be localized to one side, there will be nystagmus in addition to the deafness, tinnitus and vertigo. These symptoms recede with the disappearance of the anemia, leaving the labyrinth intact with return to normal function. If the anemia is due to chronic general causes or is the result of some slowly developing interference with the circulation in the internal auditory artery, progressive tinnitus and deafness are usually the main symptoms, although vertigo may occur. In labyrinthine anemia the deafness and tinnitus may be more pronounced in the erect than in the recumbent posture, due no doubt to change in the local blood supply.

Diagnosis.—The diagnosis in the acute form associated with general anemia offers no difficulty. The patient has his general symptoms plus those attributable to the labyrinth. In the chronic form, the progressive impairment of hearing, which tests show to be due to a lesion of the perceptive mechanism, associated with tinnitus, are common to so many conditions that the diagnosis may be difficult. One must be contented with assuming that labyrinthine anemia accounts in the best manner for the symptoms when considered with the manifestations of the disease of which it is but a part.

Prognosis.—When occurring with acute anemia or in the angioneurotic form, the prognosis is good insofar as the labyrinthine condition is concerned. Recovery with return of function may be expected. In the chronic forms the lesion is of such a nature that recovery cannot be expected.

Treatment.—The treatment is to relieve the general anemia of which it is a part. In the angioneurotic patients the bromides and quinine are of value. Bromides may be given for the tinnitus. Amyl nitrite has been suggested. Patients have been known to obtain relief from the tinnitus and other subjective symptoms by inhaling a few drops.

HYPEREMIA OF THE LABYRINTH.

Labyrinthine symptoms may be caused by hyperemia. This occurs in the early stage of serous and suppurative labyrinthitis, but outside of these diseases a hyperemia may exist which is not a part of an inflammatory process.

Causation.—In postmortem examinations in patients having acute otitis media occurring in the course of scarlatina, diphtheria and typhoid fever, hyperemia of the labyrinth has been found, so any of the acute infectious diseases may be the cause of the disease under discussion. It may also be caused by any condition which interferes with the return flow of blood from the brain, as jugular resection or tumors in the neck. Tumors in the region of the internal auditory meatus by pressing upon the veins may also act as a cause. The labyrinthine hyperemia, if long continued, may result in changes, but usually does not do so.

Symptoms.—The symptoms are tinnitus, deafness, vertigo and a feeling of fulness in the ears and head. These symptoms may exist associated with the clinical picture of cerebral hyperemia of which it is but a part. The cheeks may be flushed, the auricles red, and at times there may be found an increase in the blood supply of the fundus of the external auditory meatus.

Diagnosis.—The labyrinthine symptoms occurring in acute middle-ear processes, while possibly due to this condition, should be looked upon with suspicion as being much more probably due to some of the inflammatory complications which occur in the labyrinth. If there is evident cerebral hyperemia and labyrinthine symptoms occur and pass away with the subsidence of the condition, leaving the organ functionating normally, it is fair to assume that the condition affected the labyrinth also. It must, however, be borne in mind that hyperemia of the brain may produce vertigo, perhaps also tinnitus and deafness by involving the eighth or its central connections. Politzer claims that the diagnosis can only be

made when otoscopic examination shows hyperemia of the fundus of the external auditory meatus.

Prognosis.—The prognosis is as a rule good. In fact, if permanent change in function is produced, one would hesitate before believing that the process had been one of hyperemia pure and simple.

Treatment.—The treatment varies with the causes. These should always be found and relieved if possible. The patient should be put to bed. The head, to which cold applications are applied, elevated. Hot applications to the extremities. Calomel followed by a saline may be useful. The bromides should be administered in full doses. In very severe types local or general abstraction of blood may be advisable.

OTITIS INTERNA.

Definition.—The term otitis interna may be applied to processes which affect the inner ear and are not secondary to the purulent middle-ear inflammations. The term is not to be considered as indicating the nature of the process, but as a convenient one under which to discuss a certain type of case. Patients are continually presenting themselves who are profoundly deaf. Functional tests locate the lesion in the perceptive apparatus. Examination of the drum membrane fails to reveal any change which indicates either present or past suppuration. The history is that the patient suffered from one of the infectious diseases upon recovery from which deafness was found to be present, or the child had a fever of unknown nature which left him deaf. It is possible that in some of these patients there was a suppurative middle-ear process which left no apparent changes, to which there occurred a secondary serous or purulent labyrinthitis, which is responsible for the loss of function. Such a process is not, however, in the habit of exhausting itself and leave no evidence which can be seen upon inspection of the drum membrane. So it is assumed that there was

an inflammation of the labyrinth independent of middle-ear involvement or some pathological process in the acoustic and vestibular nerves, either in the internal auditory meatus or at the base of the brain, to account for the symptoms.

Etiology.—The most common cause of the disease is epidemic cerebrospinal meningitis. During the last ten years many patients have come to the clinics attributing their deafness to this disease. Following the epidemic of 1904–5, they were especially common. Not infrequently the condition was due to measles, mumps, scarlet fever, diphtheria, and less often influenza. At times as far as can be ascertained it is due to a fever of unknown nature. Whether or not the labyrinthine inflammation is the primary process to which the fever is due it is impossible to state. But the fact that even purulent labyrinthitis is often an afebrile disease, and when there is fever it is very moderate, inclines one to the belief that the disease under discussion is not the cause of the fever but the result of the process which produced the rise of temperature.

Pathology.—In autopsies performed upon patients deaf from epidemic cerebrospinal meningitis, the most common condition present is an inflammation of the labyrinthine structures, if the examination is made soon after the onset of the inner-ear disease. Later there is formation of connective tissue and new bone. At times the lesion consists in inflammatory involvement of the eighth nerve with degeneration of the structures within the labyrinth. Goerke[1] in fifteen cases found the infection extended from the meninges to the labyrinth through the aqueductus cochlea three times, the aqueductus vestibuli once, and through the internal auditory meatus eleven times.

In diphtheria the symptoms are believed to be due to a toxic neuritis of the eighth nerve. Alagna[2] has produced

[1] Deutsch. Otolog., 1906.
[2] Zeitschrift für Ohren., Bd. 59.

degeneration of the spiral ganglion, in the modiolus of the cochlea, in animals by the injection of diphtheria toxin. Lewin[1] found degenerative changes in the eighth nerve and its peripheral ganglia in a considerable proportion of patients dying of diphtheria. In measles and scarlatina the process is either a labyrinthitis or a degeneration of the eighth nerve. When a labyrinthitis occurs in measles or scarlatina without middle-ear involvement, it is metastatic in nature. Considerable discussion has taken place as to the nature of the lesion in mumps. There seems, however, to be no very exact conception of its nature. It is probably similar to the processes which occur in the testicles or ovaries in this disease. A number of autopsies have been reported in which labyrinthitis was secondary to non-epidemic purulent meningitis. The possibility of this occurring has been alluded to under Suppurative Diseases of the Labyrinth.

Symptoms.—The symptoms at the onset of the involvement of the labyrinth or eighth nerve, occurring as it does in a patient who is very sick with the disease of which this involvement is a complication, may be overlooked by the attendant who, perhaps, is not on the alert for their occurrence. Then again, the patient may be unconscious and the development of the symptoms cannot be detected. In epidemic cerebrospinal meningitis the intensity of the disease seems to have nothing to do with the occurrence of deafness. Very severe types may recover without aural involvement, while the abortive types, in which the general symptoms are more mild, may develop aural complications. The symptoms when they may be observed are profound deafness, nystagmus, vertigo, nausea and vomiting, and if the patient attempts to walk, an unsteady gait. The attendant upon patients suffering with the infectious diseases should be on the alert for these symptoms, for until he is impressed with their significance and especially with

[1] Zeitschrift für Ohrenheilk., 1913, lxvii.

the importance of examining the labyrinth in all patients who die after manifesting them, knowledge of the pathological basis of this type of labyrinthine disease will make but slow progress.

These patients come under the observation of the aurist suffering with profound deafness. Tinnitus is but seldom a symptom as the patients are mostly children who rarely complain of subjective noises. One may syringe the ear with cold water and produce no nystagmus or vertigo. If the disease is bilateral, one may rotate them without producing nystagmus. This is the rule, although at times there is some response to these tests, rarely being equal to normal. It would seem that for the acoustic reaction to be lost (deafness) while the static reactions were present, it would be necessary for the lesion to be in the labyrinth and affect the cochlea while the canal system escapes. This is the more rational explanation of deafness without loss of vestibular function. If one could examine the temporal bones of some of these patients who have had thorough examinations during life, the question might be settled. It is conceivable that a toxin might have a selective action on the cochlear component of the eighth nerve and produce deafness without at the same time destroying the vestibular function. Quinine and salicylic acid produce tinnitus and deafness without, in the great majority of instances at least, producing vertigo. That any coarse lesion as a tumor or exudate could select the cochlear component seems improbable.

Diagnosis.—A patient with deafness, usually profound or absolute, which is due to a lesion of the perceptive mechanism, who presents no changes of the drum membrane indicating that a suppurative process of the middle ear has been present, and who gives a history that the deafness occurred during one of the infectious diseases or a febrile attack, may be regarded as belonging to the type under discussion.

Prognosis.—The prognosis is unfavorable. In rare instances some hearing is retained, but as a rule deafness is profound. Children under seven with binaural involvement usually become deaf-mutes.

Treatment.—During the acute stage there are no means at hand to prevent or minimize the loss of function. After this stage is passed the iodides and pilocarpine should be used for several months in the hope of producing some amelioration of the deafness. Educational exercises in lip-reading and the use of the voice should be begun as soon as possible after the attack.

DEAF-MUTISM.

Deaf-mutism is the impairment of hearing to such a degree that the child cannot acquire the art of speech, or, having acquired it is unable, on account of profound deafness, to retain it without being assisted by proper instruction.

Mutism.—In order that the child may be able to learn to speak it must be able to hear the spoken words of others, that, by imitation, it may be able to utter them. Speech is usually acquired in the second and third years of life, so if there is some lesion or anomaly of the organ of hearing which results in profound deafness before this time, the child will not learn to speak. If by disease the hearing is lost during or after this period, the child loses the ability to use what speech he had acquired, unless he is promptly taken in hand by a competent teacher. After the seventh year if the hearing is lost, more or less speech usually remains. Even if the child becomes deaf before that time and after acquiring some speech it is frequently not entirely lost, the child still possessing a very limited vocabulary, to which the mother calls attention as evidence that he is not a mute.

Etiology.—Deaf-mutism is usually divided into two forms, congenital and acquired. In the congenital form the condition resulting in deafness exists at birth, while

in the acquired the impairment of hearing is caused by postnatal disease, occurring, as has been seen, at any time before the seventh year. Statistics seem to show that deaf-mutism is divided about equally between the two forms, although in an individual instance it may at times be difficult to state whether the child was born deaf or acquired the deafness very early in life. A great deal of statistical work has been done to determine the cause of congenital deaf-mutism. There is no doubt but that heredity and consanguineous marriages are etiological factors. If congenital deaf-mutes marry, or cousins in a family in which deaf-mutism seems uncommonly prevalent marry, there is great probability of some of the offspring being born deaf. Consanguineous marriages naturally increase any hereditary tendency that may exist, and it has been found that deaf-mutism is more frequent among people or communities who encourage marriage with relatives than among those who discourage or prohibit it. For this reason isolated communities, as on islands or in mountainous districts, show an increase of this disease. Altitude seems also to be an etiological factor. In the Swiss Alps the proportion of deaf-mutes is larger than at the sea level. But in the former locality cretinism is very common and it is believed that many cases are reported as deaf-mutism, in which the dumbness is due to the mental inferiority of the patient and not to deafness at all.

The causes of acquired deaf-mutism are epidemic cerebrospinal meningitis, scarlet fever, measles, typhoid fever, diphtheria, erysipelas, chicken-pox, mumps and rheumatic fever. This by no means exhausts the list of causes to which deafness in infancy or childhood may be attributed. It may be stated that any condition which conduces to middle-ear suppurations may also be a cause, as a bilateral labyrinthine involvement in this disease often leaves the child permanently deaf. Thus adenoids become of etiological significance and many patients are brought to the clinics with profound deaf-

ness in whom the nasopharynx is full of these growths. It seems impossible to consider adenoids as directly responsible for deafness, but indirectly as favoring infection of the middle ear they may be a cause. Of the patients seen in the clinics of New York during the last ten years, the writer would place epidemic cerebrospinal meningitis first in causation, then follow closely scarlatina, diphtheria, and measles. Often it seems impossible to ascertain the cause, the mother stating that the child was all right until it had a fever, since which time she has noted the infirmity. Congenital syphilis is no doubt responsible for some cases. It is probable that its importance in etiology has been underestimated.

Pathology.—In by far the greater number of patients the lesion is in the sound-perceiving mechanism. While in some instances disease or malformations located in the conducting mechanism produce sufficient deafness to result in mutism, it is in a small minority of the patients that it does so. As autopsies are not frequently performed upon deaf-mutes in this country by observers who are alive to the importance of making a careful examination of the ear, knowledge of the pathological basis of deaf-mutism is derived largely from the work of those abroad, chiefly Mygind and Denker. Congenital atresia or absence of the external auditory canal is found during life responsible for some instances of deaf-mutism. In reports of autopsies are found instances of the following conditions: absence of the ossicles or of the incus; ankylosis of the stapes; labyrinthine fenestræ absent or occluded; cochlea imperfectly formed, either consisting of one and a half turns, the apex being a cavity without structure, or the turns not formed at all; cochlea absent; absence of Corti's organ; entire labyrinth absent; semicircular canals one or more absent or fused together and in abnormal position; auditory nerve degenerated and atrophied, internal auditory meatus contracted. These have all been found in the congenital form. Manasse has demonstrated the changes characteristic of otosclero-

sis in very young children, and Alexander believes that this disease may occur *in utero* and be the cause of congenital deafness. Intra-uterine syphilis may result in the child being born deaf. Labyrinthitis following upon middle-ear disease, occurring immediately after birth, might produce deafness and it might be impossible to state at a subsequent time that the condition did not arise *in utero*. The author operated upon a double subperiosteal abscess in an infant ten days old. Otorrheas are not infrequent in the first two weeks in life and both ears are not seldom involved. So the possibility of the disease extending to the inner ear is not a remote one. In reading the autopsy reports in the acquired deaf-mutism, one is impressed with the great resemblance of the changes to those found in the latent stage of suppurative labyrinthitis. In very many instances the cochlea and semicircular canals are filled with osseous or fibrous tissue; occasionally the contents are of a caseous nature. As these labyrinthine changes are often found associated with various cicatricial distortions in the tympanum and drum head, it is probable that an otitis media purulenta leads to an infection of the labyrinth through the oval or round windows, with a consequent inflammation which, passing into the latent stage, resulted in the deposit of bone or fibrous tissue. This is perhaps the usual way, but there is no doubt but that labyrinthitis may occur by metastasis. There seems no good reason why scarlatina should not produce an inflammation of the labyrinth as well as a nephritis, or why mumps should not do so as well as an ovaritis or orchitis.

Symptoms.—The deafness must be profound to cause mutism in an otherwise normal individual. With poor mental development a lesser degree of impairment of hearing will result in dumbness. In younger children it is difficult to determine what amount of hearing exists, and it is impossible to apply the tests by which the lesion is located in the sound-perceiving mechanism. Slapping the hands behind the child, taking care that a current of

air is not produced that will be perceived by the tactile sense, is a favorite way. If the child's attention is held in some other direction and it turns around to investigate the source of the sound, it is safe to infer that it possesses some hearing. A tuning-fork held to the ear will at times cause a brightening of the expression, showing perception of sound. When one sense is lost the others become keener to compensate for it insofar as possible. These little patients are already beginning to be very acute with the sense of sight and touch. They will take notice of any sound which produces a coarse vibration. The tuning-fork placed upon the head probably conveys to them only the vibration appreciated by the tactile sense. In older subjects one may ascertain that there is no bone conduction by the fact that they experience the same sensation when it is placed upon the olecranon or some other subcutaneous part of the skeleton. In older children and adults, it will be possible to locate the cause of the deafness as being in the sound-perceiving or conducting mechanism. As a few of these patients possess some audition, it is very desirable to determine at the earliest moment possible how much is heard, and the pitch of the tones perceived. This is best done by the Bezold-Edelman continuous tone series, and if a tone island is found of such a pitch that the human voice may be attuned to it, great assistance in teaching the art of speech is afforded. Some patients with acquired deaf-mutism give a history of vertiginous symptoms during the attack causing deafness. These are readily accounted for as the symptoms of the manifest stage of the labyrinthitis from which they suffered. Rarely this vertigo may persist for some time, but an unsteady gait is somewhat more common. Deaf-mutes are usually negative to the rotation and caloric tests, although occasionally one responds. It has been claimed that as deaf-mutes are not affected by currents of endolymph within their static labyrinths, as these cannot as a rule be produced on account of destruction of the

labyrinth, they are immune to seasickness. It is also stated that while they are able to swim on the surface as long as they can orient, or place themselves in space with their eyes, it is unsafe for them to dive beneath the water. However this may be, they are (usually) deprived of the static impulses normally derived from the labyrinth and are therefore less apt in maintaining their equilibrium under trying circumstances.

Diagnosis.—The recognition of deaf-mutism is in the majority of patients comparatively easy. It must be determined that the patient is deaf and that on account of his deafness he does not speak. This is not difficult when the time of life arrives when the faculty of speech should be acquired. Before this time of life the question resolves itself into determining whether or not the patient is sufficiently deaf to be unable to acquire the art of speech. During the first six months of life this is impossible, although it has been demonstrated that even during this period infants hear and take notice, although many do not react to sound. During the second six months the child should react to a loud sound, as of clapping the hands, ringing a bell, etc., but if they do not, one cannot pronounce them deaf. During the second year, when children are usually learning to talk, one cannot say that a child is deaf because he does not talk. Usually, however, the children readily react to sound, and if this is the case, enable one to assure the parents that in all probability the backwardness in speaking is not on account of the deafness and therefore not permanent. It should also be determined whether or not the child is mentally defective. Cases of hysterical deaf-mutism occur occasionally and attract considerable attention in the lay press, but should not cause difficulty in diagnosis.

Prognosis.—The prognosis as to the cure of the lesion causing the deafness is bad. This in most instances is permanent. Occasionally there is some improvement under treatment. Patients with a small amount of hear-

ing and those who have at one time spoken make very encouraging pupils for special instruction in lip-reading and articulation, while in those totally deaf or who have never spoken the results obtained are not so good.

Treatment.—The prevention of deaf-mutism, consists in improving the social and hygienic surroundings of the people at large, and the discouraging of intermarriage among relations, especially in families in which the condition has occurred. Congenital deaf-mutes should be discouraged from marrying for the same reason. Something may be done to prevent the lesions which produce profound deafness in children by a rational treatment of the suppurative process to which they are secondary. The adenoids should be removed and the suppurations treated along the lines already discussed. Of special value in preventing labyrinthine involvement is the establishment of early and sufficient drainage. After deafness has occurred a thorough examination is to be made and any measures which give promise of at all improving the hearing adopted. Even a slight diminution of the deafness may result in taking the patient out of the mute class, although he may still be profoundly deaf. If atresia of the external auditory meatus exists and is bilateral, the question of attacking it surgically arises. Page's operation, alluded to under Microtia, doubtless saved his patient from becoming a deaf-mute.

After a patient recovers from epidemic cerebrospinal meningitis, treatment, if begun early, is occasionally of some value. The administration of pilocarpine to the point of physiological tolerance, together with the iodides, sometimes, although rarely, ameliorates the deafness to a slight degree. It should be tried for the sake of this occasional patient. If some hearing remains, it must be used and encouraged. This may be done by the use of the various aids to audition, such as conversation tubes, trumpets, etc., and also by pitching the voice if possible in consonance with any tone islands which it has been ascertained that the patient possesses. By this means

it may be possible not only to retain the hearing power, but also prevent the occurrence of mutism. The retention and improving of audition, when possible, is the best treatment for mutism. When impossible the patient must be taught to communicate either by signs or speech. The latter is unquestionably superior. It originated with a Spanish monk, Pedro de Ponce, but is nevertheless called the German method, and when attainable is better than the former, sometimes called the French method. It is found that those patients who possess some hearing, however little, and those who at one time were able to speak, acquire very much more proficiency both in lip-reading and in the art of speech than those who do not have this advantage. Moreover, the length of time that the faculty of speech has remained unused has a direct bearing also. The duty of the aurist is therefore clear and imperative, and that is to place any patient who has once been able to speak and has lost this faculty through deafness under the immediate care of a competent instructor. While waiting for the child to be "old enough" valuable time is sacrificed, the loss of which is irreparable. A child who has never spoken and is absolutely deaf should be put under instruction at least as young as three years.

The subject of the method of instruction, as well as many other phases of this interesting subject, cannot be discussed within the limits of a book of this character.

CHAPTER XIII.

GENERAL AURAL DISEASES.

SYPHILIS OF THE EAR.

External Ear.—Primary syphilitic affections of the external ear are rare, but several have been reported. They have been due to the patient being bitten by a syphilitic subject, or the virus being conveyed upon some intermediate substance as a towel, etc. Secondary affections of the auricle occur as a part of a general macular, papular or pustular eruption.

Condylomata.—Children may have condylomata on the posterior surface of the auricle, in the fold between this structure and the head, where the parts are often moist and macerated from intertrigo. Condylomata may develop in the meatus, especially if there is a middle-ear discharge. They occur as red or a grayish-red, ragged, warty excrescences, springing from the canal walls, usually at the entrance to the meatus, but they may invade the deeper part or even the osseous meatus as well. They are usually painless at first, but as they become more developed they may cause pain, especially upon movement of the auricle. There may be deafness and subjective noises from obstruction of the lumen of the canal. They either heal by resolution or break down and form ulcers. Condylomata of the meatus disappear with antisyphilitic treatment, but may leave more or less atresia of the canal. Local treatment consists in keeping them dry.

Gumma.—Gumma of the auricle and canal is occasionally seen, and is usually associated with a similar process in the middle ear. It breaks down, forming an ulcer with a deep base and elevated margins. If one is

alive to the possibility of the condition being specific, proper measures for diagnosis and treatment may be taken.

Membrana Tympani.—A small gumma may be formed which may break down, producing a small ulcer on the surface of the tympanic membrane, although the process is more apt to produce a perforation or total destruction of the membrane. The author has had under his observation a patient with a small granulating surface upon the tympanic membrane due to breaking down of a gumma. A thin discharge of a purulent nature was present. Inflation demonstrated the absence of a perforation. Wassermann reaction strongly positive and history of syphilis (primary lesion occurring four years previously) was present. The condition proved quite stubborn, but finally yielded to antisyphilitic treatment (mercurial injections) and applications of strong solutions of nitrate of silver.

Middle Ear.—Suppurations in the middle ear may be caused by extension of a specific process in the nasopharynx, through the Eustachian tube to the tympanum. It may be difficult at times to determine whether one is dealing with a syphilitic otitis media or a simple process in a syphilitic subject. Rapid destruction of the membrane with a milder degree of pain may be expected if the process is specific. Occasionally such a process invades the mastoid, or the mastoid may be the seat of a gummatous infiltration not secondary to middle-ear syphilis. After operation if the nature of the process is not recognized the bone may become progressively involved and break down. The process may thus extend into the occipital bone, into the squama, and into the depth of the temporal bone, producing labyrinthitis and facial paralysis. The meninges may become thickened and agglutinated with the cerebral cortex. This condition will, as a rule, rapidly clear up if the nature of the process is recognized and the patient put upon vigorous antisyphilitic medication.

Inner Ear.—Syphilis not infrequently involves the labyrinth and eighth nerve.

Pathology.—Autopsies, which have been performed upon patients who had presented symptoms fairly attributable to syphilis of the inner ear, have shown the following conditions: thickening of the endosteum of the vestibule; stapediovestibular ankylosis associated with small-celled infiltration of the tissue between the membranous and bony labyrinths, of Corti's organ and of the membranous canals; degeneration of the spiral ganglion with small-celled infiltration and extravasation of blood between the fibers of the acoustic nerve; narrowing of the internal auditory meatus and deposit of new bone in the labyrinthine spaces; round-celled infiltration in one or both acoustic nerves. Gummatous inflammation of the dura in the internal auditory meatus, or of the nerve sheath, may take place. Syphilitic meningeal processes may occur on the posterior surface of the petrous pyramid and lead to involvement of the eighth usually in conjunction with other cranial nerves. The eighth nerve, on account of its consistence and its location in a canal where it may be subjected to pressure, is one of the most vulnerable of the cranial nerves.

Course and Symptoms.—The eighth nerve or labyrinth is most frequently involved in the tertiary stage of syphilis, or on the border of the secondary and tertiary stages. That it may occur early in the disease and does so much more frequently than was formerly supposed, has been demonstrated through the impetus given to the study of syphilis by the discovery of the Spirocheta pallida, the complement-fixation reaction and the use of salvarsan. Otto Meyer[1] found in the records of the Grazer ear clinic between 1896 and the inception of salvarsan medication, 65 cases of disease of the inner ear, of which 20 per cent. began within the first year after infection. Instances have been reported in which the inner-ear involvement has occurred as early as two months after the initial

[1] Weiner klin. Woch., 1911, No. 11.

lesion, so there is really no time in the secondary or tertiary stages that the specific patient is exempt.

The symptoms are deafness, tinnitus and at times vertigo and nystagmus. The deafness usually comes on rather suddenly, that is, it develops and becomes profound within a few days, although there are occasionally instances of its slower progress. Tests show this deafness to be due to lesion in the perceptive mechanism, A. C. > B. C., B. C.– and Weber to the good ear. One peculiarity which has been noticed about the tuning-fork tests is the marked shortening of the bone conduction. Instead of being one-half of air conduction with the 256 D. V. fork, as occurs in many labyrinthine diseases, in syphilis of the inner ear this ratio may be changed so that bone conduction is much less than half of air conduction. In eliciting bone conduction in patients with very marked difference on the two sides, when the fork is applied to the deafer ear, it may be heard through the head on the side of the better ear and give rise to the impression that B. C.> A. C. (bone conduction is greater than air conduction) as the air conduction is very much shortened or even lost, that is, the patient does not hear the fork through the air but appears to through the bone, and this in an involvement of the perceptive mechanism, which of course is impossible. One will not make this error if he takes pains to ascertain on which side the patient hears the fork when applied to the mastoid. The bone conduction for the higher-pitched forks being naturally shorter than that for the 256 D. V. is lost first, that is, the 512 D. V. or 1024 D. V. forks will show loss of bone conduction in milder degrees of impairment of hearing than will the 256 D. V. or the lower forks. While much has been made of these bone-conduction tests with a series of forks, the writer is satisfied that as a rule one derives about as much knowledge of the kind and location of the lesion if he uses a good fork of 256 D. V. and accustoms himself to rely on its findings. Tinnitus is a frequent symptom and may be very distressing. Some patients

develop their tinnitus and deafness without vertigo, or this may be a very prominent symptom in the beginning of the attack.

Vertigo is generally associated with nystagmus, especially if one side only is affected or to a greater degree than the other. With the vertigo there may be vomiting and a tendency to fall, in fact all of the symptoms of disharmony described under Purulent and Serous Labyrinthitis. These symptoms usually pass away, but the deafness remains. Unless prompt treatment is instituted, it usually becomes profound. When these patients come under observation after a lapse of one or more months, the vestibular function is usually found destroyed. They do not react to the caloric or to rotation tests if the disease is bilateral. If unilateral, the rotation test may show compensation or a negative reaction. In rare instances the hearing may be nearly or quite destroyed and the static labyrinth still remains active.

Diagnosis.—Deafness occurring in a syphilitic subject (with or without vertigo and nystagmus) which tests show to be due to a lesion of the sound-perceiving mechanism, is presumably syphilitic in nature. If the bone conduction is less than half of air conduction, with the 256 D. V. fork, it tends to strengthen the supposition. The possibility of such a process being other than luetic in nature should be disregarded and treatment instituted along antisyphilitic lines. In determining whether or not the patient is luetic, the history is carefully taken. No matter how soon after the initial lesion the symptoms present, they are still to be regarded as indicating a process of specific nature. Due regard must be paid to the complement-fixation tests, although it is possible that the disease may be specific and that they should be negative. At times when the blood gives a negative reaction, if the cerebrospinal fluid is used a positive reaction may be obtained. So if there is doubt, the Wassermann reaction should be tried on this fluid obtained by lumbar puncture. It is often impossible to obtain a history of hereditary

syphilis, but usually the subject of this form of lues presents its stigmata. There may be interstitial keratitis or evidence of its having been present, or the well-known Hutchinson's teeth. The characteristic of these incisors is that they are peg-shaped, that is, broader at the gum than at the cutting edge, whereas normal incisor teeth are broader at the cutting edge than at the gum. This condition of the incisors, with interstitial keratitis and deafness is known as Hutchinson's triad, and is very characteristic of hereditary syphilis; although the aural involvement is less frequently seen than the ocular. To determine whether the lesion is in the labyrinth or involves the eighth nerve or its central connections, may be impossible. If the facial is also involved and no reason exists for believing that this is due to some process peripheral to the fundus of the internal auditory canal, it is safe to assume that the eighth together with the seventh is involved in this canal. If in addition to the facial the sixth, perhaps also the fifth, is implicated, the lesion may with a fair degree of certainty be located on the posterior surface of the petrous pyramid in the cerebellopontine angle.

Prognosis.—The prognosis as to return of function is not good. If early treatment is adopted, the improvement at times may be truly remarkable, but the otologist usually sees these patients after irreparable injury has been done to the labyrinth or nerve, so that a moderate improvement is the most that may be expected. The patients who have inherited syphilis are usually the most hopeless, but they may at times show some improvement.

Treatment.—The patient should be put upon vigorous constitutional treatment at the earliest possible moment. This is especially necessary if he presents early in the involvement of the inner ear. He should be given mercury and perhaps also the iodides at once. On this there is now practical unanimity. Whether or not he should also receive salvarsan has been a subject of much discussion.

Salvarsan and Eighth Nerve Lesions.—After the use of salvarsan in the treatment of syphilis became more or less general, it was found that patients began to present themselves early in the disease and at varying times after the administration of salvarsan with involvement of the eighth and other cranial nerves. It was claimed on the one side that this neurorecurrence, as it was called, was a result of the syphilis itself and had nothing to do with the salvarsan, while the other side believed that it was due in a greater or less degree to the administration of this remedy. It was shown that syphilis of the inner ear was much more common in the early stages of the disease in the antesalvarsan period than was generally supposed. Moreover, many of these cases were reported in which the aural symptoms following a dose of salvarsan were improved or relieved by one or more administrations of the remedy, or by mercurial treatment. Still many patients did not improve. The known effect of 605 upon the optic and acoustic nerves when administered for sleeping sickness caused many to believe that the arsenic in "606" might be responsible for the neurorecurrences. Without discussing the subject at length it may be stated that the opinion prevails, that while it is possible that the arsenic may cause involvement of the eighth, it does not commonly do so but that the condition is due to the growth of the spirochetæ which have escaped the action of the salvarsan. Nichols'[1] theory is probably true. He believes that while these organisms are in the main killed by the salvarsan, this remedy acts so thoroughly that no natural resistance is established as when mercurials are used, and a focus which may exist in the nervous system finds its development unopposed after the elimination of the salvarsan. The practice at present considered safe is as follows: if the inner-ear involvement occurs in the early stages, salvarsan followed by full doses of mercury should be given; if

[1] Journal Amer. Med. Assn., March 2, 1912,

in the later stages, it is well to give mercury for a short time before "606" is administered, and continue the mercurial treatment for some time afterward. For the neurorecurrences which follow the use of salvarsan, mercurial treatment is the best prevention and, associated with salvarsan, offers the best prospect of cure once the condition is present.

TUBERCULOSIS OF THE EAR.

External Ear.—Tuberculosis of the auricle may exist as one of the forms of lupus. The consideration of these conditions is within the province of the dermatologist, as they rarely present any phase of interest to the otologist.

Middle Ear.—An otitis media or mastoiditis may occur in a tubercular subject and be due to infection with other pyogenic organisms, or the process may be caused by the tubercle bacilli. These latter organisms may be found in the aural pus or careful search may fail to show them, even when the clinical picture seems characteristic.

Causation.—The disease may be primary, which is considered of uncommon occurrence, or secondary to tuberculosis in the lungs, lymph nodes, nasopharynx or osseous system. In the act of coughing, the tubercular pus is often thrown with force into the nasopharynx and it would seem to be easy for it to reach the middle ear through the Eustachian tube. This, no doubt, is frequently the case and is the usual mode of infection in those tubercular middle-ear processes which occur in a comparatively late stage of pulmonary disease, when expectoration of material swarming with bacilli is the rule. Adenoid vegetations or tonsils which, as is well known, are frequently the seat of tubercular foci, seem favorably located to produce middle-ear infection either through the lumen of the Eustachian tube or through the submucous lymphatics situated in this structure. In general miliary tuberculosis the infection takes place through the lymphatics or blood stream. When diseased

lymph nodes are present in the neck, the question whether they are secondary to the aural process or the converse may arise. It has been shown that many of the acute suppurations of the middle ear in children are of a tubercular nature.

Pathology.—The examination of the mucous membrane of the middle ear shows round-celled infiltration, giant cells, groups of tubercle bacilli with foci of destruction of the membrane. Tubercular nodes are found in the granulations. The process has a marked tendency to affect the bone, producing necrosis, caries or softening. The ossicles are often destroyed and the labyrinth or Fallopian canal invaded. The mastoid, especially if of pneumatic type, is invaded and the inner table broken down, producing extradural collections of pus.

Symptoms.—As to the symptoms produced by tubercular invasion of the middle ear and mastoid, two types are found. In the first the clinical history very much resembles the ordinary acute suppurative otitis media, and the second is characterized by the absence of reactionary symptoms. In the former type the onset is acute and the nature of the process may not be suspected until the occurrence of great destruction of the membrana tympani, multiple perforations, or the enlargement of the neighboring lymph nodes calls attention to its probable nature. The rapid formation of granulations in acute otitis media often occurs in tubercular processes. Facial paralysis and labyrinthine complications are more frequent also. Next to cholesteatoma, tuberculosis is the most common cause of fistula of the labyrinth. It is also not rarely responsible for diffuse suppurative labyrinthitis with sequestration of a greater or less part of the labyrinthine capsule. In the chronic type the discharge appears without pain. There may be more or less discomfort and feeling of stuffiness in the ears, but usually the patients do not come under observation until the ear is discharging and when questioned give no history of pain or discomfort, but come for the discharge

and deafness which is present. From the fact that this type of tuberculosis of the ear is more apt to be recognized on account of this characteristic absence of pain, it has been erroneously stated that tubercular otitis media occurs without pain. An accumulation of pus within the ear sufficient to produce rupture of the drum membrane will produce pain. In the chronic type the membrane is involved and breaks down before the tension within the tympanum becomes sufficient to cause pain.

Signs.—If a patient with the painless type comes under observation early, the appearance of the drum membrane is quite characteristic. It is of a dull red or pink color, the luster is diminished and the membrane seems thickened. At one or more points are areas which appear of a yellowish color. These are the seat of tubercular nodules in the process of breaking down. In some instances this goes forward slowly and these areas are not converted into perforations for a month or more, in others the time required is less. If the ear is inflated, moisture will usually be found present. During this time the patient has deafness and perhaps tinnitus but no pain.

When perforations are present they may appear not unlike the ordinary ones or their margins may be thickened and apparently everted, the thickened edges having a grayish or yellowish color. If the perforation is a large one, the internal tympanic wall may be seen to be covered with granulations, among which bare or loose pieces of bone may at times be detected with a probe. At times caseous material may be found in the middle ear and the mastoid at operation may be found to be filled with the same cheesy material.

Diagnosis.—The diagnosis in patients in whom the onset is painless may not be difficult. This feature alone calls one's attention to the probable nature of the process. If upon examining the drum membrane multiple perforations are found or a single one with characteristic margins, one is justified in assuming that the process is

a tubercular one. If the patient also has tuberculosis of the other parts, it substantiates the diagnosis. If the patient is seen before the tympanic membrane ruptures in this type of case, its appearance is sufficiently characteristic to justify a diagnosis, especially if pulmonary tuberculosis is present.

In the acute types which develop with reactionary symptoms the nature of the process is often not made out or even suspected at first. The author has been impressed with the considerable number of patients who have apparently developed pulmonary tuberculosis after the mastoid operation. The middle-ear process and complicating mastoiditis have given no clinical manifestations which have attracted attention to the probability that the process was tubercular, and yet, during the healing of the mastoid wound, the patient is found to have pulmonary tuberculosis, which gave no symptoms directing attention to its presence prior to the otitis media and mastoiditis for which operation became necessary. The possibility naturally suggests itself that the pulmonary disease in such instances may have been secondary to the process in the middle ear and mastoid.

An aid to diagnosis is the von Pirquet reaction. This, if positive in a child under two years of age, is highly suggestive that the process is tubercular, especially if no other process of this nature is present. The most certain method of making the diagnosis in doubtful cases is to submit tissue excised from the middle ear to microscopic examination. If tubercular bacilli are found in the aural pus in fair quantity, one would be justified in assuming that the process was tubercular. It has, however, been found that they may not show in smears or grow in cultures of aural pus from processes evidently tubercular. Inoculating a guinea-pig has sometimes demonstrated their presence in such instances. Sometimes the pig dies from the effect of the pyogenic organisms which exist in the pus injected, and the experiment is without value. If it proves successful, the result is not known until valuable

time has been lost. A method of obtaining bacilli from smears and cultures is described by Cocks and Dwyer.[1] It is as follows:

"The discharge was obtained in as large a bulk as possible in a small quantity of normal salt solution, the latter being used in an amount just sufficient to wash out the pus. The water used in making up the salt solution was freshly distilled each day in order to be sure that none of the acid-fast organisms present in tap water or in old distilled water could vitiate our results. This discharge was then treated with an equal amount of 15 per cent. antiformin, and the whole was allowed to stand for a varying period, depending upon the consistency of the mixture, etc. It was then centrifugalized, and the precipitate was washed in order to remove the excess of alkali. Smears were then made from the precipitate and stained by the Ziehl-Neelson and Pappenheimer method. In this way we were able to demonstrate the organism with reasonable certainty. By this method, no matter how much care is exercised, there is always an element of uncertainty in morphological diagnosis alone, as in many of the old chronic suppurating ears the acid-fast epithelioid flakes are apt to be mistaken for tubercle bacilli. These flakes are present in a large proportion of chronic cases.

"In the series reported below, an effort was made to isolate the organisms from the discharge and cultivate them directly upon special media, so that there could be no question as to the diagnosis. Thus, animal inoculation was eliminated and much time was saved. This was made possible by the use of Petroff's media, a full account of which appears in the *Journal of Experimental Medicine*, January, 1915. During the summer of 1914, Petroff, working in the bacteriological laboratory of the College of Physicians and Surgeons, Columbia University, devised

[1] Laryngoscope, St. Louis, March, 1915.

a specific medium, which we have used successfully for cultivating the tubercle bacillus. Thus far we have tried this method on the discharge from thirty chronic suppurating ears, and have isolated tubercle bacilli in three cases. The three positive cases were found in making the routine examinations of children who presented themselves at the Manhattan Eye, Ear and Throat Hospital, suffering from chronic purulent otitis media.

"The method we employ is somewhat different from Petroff's. It is really a combination of Petroff's method of isolating from sputum and his method for feces. This modification was rendered necessary by the large number of spore-forming organisms often present in chronic otitis. Our technic is as follows:

"After obtaining the aural discharge in wide-mouth bottles, it was immediately saturated with sodium chlorid and allowed to stand for from half an hour to an hour. At the end of this time, the bacteria are found floating on the surface. This floating film is then collected with a deflagration spoon in a wide-mouth bottle, and an equal volume of normal sodium hydroxid added. The mixture is shaken well, and left for digestion in the incubator at 37° C., for one or two hours, or longer, care being taken to shake it every half-hour. The mixture is then neutralized to sterile litmus paper with normal hydrochloric acid, and the sediment is inoculated into several test-tubes. Growth usually occurs in from fifteen to thirty days."

Prognosis.—The prognosis in tuberculosis of the middle ear is as a rule unfavorable. When it occurs in advanced pulmonary tuberculosis it may not materially contribute to the fatal outcome. In some instances, however, it leads to a fatal issue by producing labyrinthitis and meningitis. The possibility of the process being primary and acting as a focus of infection should also be borne in mind. If the patient's general health improves, the local ear process sometimes gets well. After the mastoid operation the wound may heal, but it frequently fails to do so.

Treatment.—In primary tuberculosis of the mastoid and middle ear, free opening of the bone and removal, so far as possible, of all diseased tissue is indicated. In children with complicating enlarged lymph nodes, a like procedure should be undertaken. In the chronic type occurring in pulmonary tuberculosis the condition of the lungs largely determines whether or not operation is advisable. The process in the temporal bone may be of such a nature as to menace life, and must then be dealt with surgically. The bone destruction may progress without producing marked symptoms and the insidious nature of the process must be borne in mind. If the pulmonary condition is quiescent, the general nutrition good, and the local aural disease seems to be hindering convalescence, an operation is justifiable. If, on the other hand, the disease in the lungs is getting worse and the disease in the temporal bone seems merely an incident, operation will only hasten the end. An item is the effect of the anesthetic on the lungs. If it becomes necessary to perform an operation on a patient with pulmonary involvement, ether should not be administered. The safest anesthesia is by the "gas-oxygen" method, which seems to have less unfavorable effect on the lungs. The general health of these patients with tubercular ear processes demands special attention. Their resistance and nutrition should be increased by all means possible.

Inner Ear.—Tuberculosis of the inner ear usually assumes one of the forms of labyrinthitis which are secondary to middle-ear suppuration, either sequestration of a part of the labyrinth leading to a circumscribed inflammation, or the death of the bone, resulting in diffuse purulent labyrinthitis. These sequestrations of the labyrinthine capsule may be of sufficient size to embrace nearly the entire labyrinth, exposing the carotid artery, and leading in extreme instances to almost total destruction of the petrous pyramid. The facial nerve in such a process would naturally be destroyed, and it may be involved in the lesser ones also. The treatment is

to remove the sequestrum, drain the labyrinth or adopt other operative procedures, according to the indications which are discussed under Labyrinthine Diseases Complicating Purulent Otitis Media.

TRAUMATIC LESIONS OF THE EAR.

External Ear.—Traumatic lesions of the auricle sometimes occur. The contusions have been alluded to as producing hematoma. Incised and lacerated wounds are treated upon general surgical principles. Care must, however, be taken to prevent infection on account of the danger of perichondritis. It may be necessary before closing a lacerated wound to dissect out some of the cartilage to prevent it being exposed during the healing process. Its removal minimizes the danger of perichondritis.

Middle Ear.—A fracture of the base of the skull may invade the tympanic cavity without at the same time involving the labyrinth. If the membrana tympani is ruptured, blood appears in the meatus. The mere presence of blood does not indicate that the tympanum is involved and the membrane ruptured, as fracture of the external auditory canal or simple laceration of its membranous portion may cause hemorrhage from the ear. If the lesion involves the tympanum and the membrana tympani does not rupture, the blood accumulates internal to this membrane, producing a bluish appearance. The question may arise whether or not the membrane should be incised to allow the escape of the blood. One usually hesitates before doing this on account of the danger of introducing infection, but if pain or signs of inflammation are present it becomes one's plain duty to do so, and to treat the patient as for a purulent otitis media. If such a patient should develop symptoms of a mastoiditis, one would operate early, as the possibility of infection entering the cranial cavity through the fissure produced by the

injury would cause one to assume no added risk by delaying operation.

Inner Ear.—The fissure may extend through the labyrinth involving the terminal apparatus of the eighth nerve, or through the internal auditory meatus affecting the trunk of this nerve. Nor does it seem necessary that the bone should be fractured to produce a serious lesion of the nerve or labyrinth, as at times the concussion is sufficient, the mere jarring seems to so change the delicate structures that loss of function results.

Symptoms.—Lesions of the inner ear as a result of fracture of the base usually give few symptoms, as the patient is unconscious from the injury. If, however, this is not the case, there will be deafness, tinnitus, vertigo, and nystagmus, of which the deafness, perhaps the tinnitus also, is usually permanent. In concussion of the labyrinth vertigo and nystagmus may be present during the early stages in addition to the deafness and tinnitus.

Diagnosis.—The otologist usually sees these patients at some time after the injury when the vestibular symptoms have subsided and the deafness, and perhaps also the tinnitus, remains. It is usually not difficult to satisfy oneself that the labyrinth or eighth nerve has been injured, by the history of injury and deafness due to lesion of the sound-perceiving mechanism, probably also loss of caloric reaction and change in the rotation reaction.

Medicolegal.—In medicolegal cases it is necessary to determine that the patient's hearing was good before the injury. A claim for damages may be made and loss or impairment of hearing attributed to an injury which the claimant alleges he has received. If it can be proved that his hearing was good before the injury, while after it he suffered from deafness which can be demonstrated to be due to a lesion of the perceptive mechanism, it is a reasonable inference that some lesion of the labyrinth or nerve was caused by the injury.

Prognosis.—The prognosis in those patients who show total loss of function is unfavorable. If the impairment

of hearing is less, it becomes more favorable. However, it is always more or less uncertain.

Treatment.—During the first stage after injury and in the presence of symptoms referable to lesion of the static mechanism, rest in bed with the head elevated and cold applications to the head should be ordered. A certain number of fractures of the base cause death by meningeal infection introduced through a perforation in the drum membrane. Hence, the ear should be syringed with a bichloride solution and boracic acid insufflated. For the after-treatment the iodides may be given. Strychnine has proven of value in the patients with "concussion of the labyrinth." Pilocarpine should be given a trial for a month or so.

Mauthner[1] has written at some length on traumatic affections of the inner ear.

[1] Arch. für Ohrenheilk., lxxxvii, 146.

CHAPTER XIV.

TINNITUS AURIUM.

The subjective sensation of sound is a symptom which has been constantly alluded to in these pages as tinnitus. It is an aural symptom of very common occurrence, and is usually associated with deafness. The term subjective noises is usually applied to the sensations of sound which have no physical basis outside the patient. It is necessary that the patient recognize this fact, otherwise his belief that the noise is produced outside would constitute an hallucination. If the sound can be perceived by the examiner, it is called an entotic noise. This term is evidently an incorrect one, as in many instances the sound does not arise in the patient's ear, but is due to some abnormality in the intracranial circulation. Frequently the patient, while recognizing the subjective nature of the noise, cannot resist the belief that the sound ought to be heard by others also. One is not infrequently asked if he cannot detect the noise through the auscultation tube. If this is possible, which is rarely the case, it is called an entotic noise.

Variety.—The varieties of sounds described by patients is endless. "Hissing as of escaping steam," "humming," "ringing," "roaring," "clanging," "buzzing as of a bee," or some other insect, are some of the terms applied. The sound may remain more or less continually in one pitch, or may vary from day to day. Sometimes a musical tune will be heard, or a short strain repeated over and over again. The noise may be continuous or stop for intervals. Not infrequently it is of a pulsating character, occurring with greater intensity at the time of the cardiac systole.

Causes.—Occurring as they do in diseases of the auditory nerves, of the labyrinth, and of the conductive mechanism, it is evident that more than one cause may operate to produce these perversions of hearing. An irritation of the auditory nerve may produce the subjective sensation of noises. This may be due to disease of this structure, or from the effect of various substances which seem to have a selective action upon the nerve such as quinine, salicylic acid, and the toxins generated in disorders of the digestive organs. It is stated by some authors that they are always caused by irritation of the auditory nerve or its terminal filaments in the labyrinth. While this may be true in the majority of instances, it cannot be conceded in every case. If one occludes the external auditory meatus with the finger, there is immediately produced a rumbling sound. This is not produced by irritation of the auditory nerve or its terminal filaments. The explanation for this phenomenon doubtless accounts for the production of tinnitus in many instances in which it is present as a symptom of disease in the sound-conducting mechanism. The noise produced by the circulation of the blood in the labyrinth and the bloodvessels adjacent to it is below the threshold stimulus for the perceptive mechanism, and therefore is not heard by the normal ear. It is known that with disease of the conducting mechanism, vibrations of the labyrinthine fluid produce more effect upon the acoustic apparatus. This accounts for the increased bone conduction which is present in these diseases. The labyrinthine fluid is with more difficulty set in vibration by aerial conduction on account of the disease, but if caused to vibrate by conduction through the bone, the fork is heard louder and longer on account of the disease. It is evident that the sound produced by blood circulating in the labyrinth and vessels adjacent to it produces an effect which in the normal state is inaudible, but with the increased perception for a given vibration of labyrinthine fluid, which has been shown to occur in diseases of the conducting mechanism,

it rises into consciousness and is perceived as a noise. This accounts for the sound perceived when the finger is inserted into the ear, and also doubtless for many subjective noises. Strictly speaking, sounds produced in this manner have a physical basis, while those produced by pathological irritation of the acoustic nerve arise solely from a stimulation of a non-sonorous character which is interpreted as sound. Increased labyrinthine pressure may also cause tinnitus. This doubtless acts by producing some disturbance of the organ of Corti which is interpreted as sound.

Prognosis.—The prognosis of subjective noises depends largely upon the disease of which their occurrence is a symptom. In the suppurative middle-ear diseases they are more apt to disappear than in the hyperplastic form of chronic catarrhal otitis media. In tubal catarrh, tubotympanic congestion and the exudative type of O. M. C. C., the prognosis is not so bad. In otosclerosis they frequently persist even at times when deafness is profound. In nervous subjects tinnitus has led to suicide.

Treatment.—The treatment of noises has been detailed under the various diseases in which this symptom occurs. It is desirable to keep the patient's mind, as much as possible, from concentrating upon the tinnitus. Constipation is able to keep up the tinnitus or intensify it. Increased arterial tension is at times responsible for it, so the blood-pressure should be taken and if high, measures adopted to reduce it. In severe cases when death becomes preferable to enduring the noise longer, the possibility of relief by surgical measures naturally suggests itself. While these are more or less disappointing, they may be tried after acquainting the patient with the risks and probable result. Dr. Dench divided the auditory nerve at the internal auditory meatus in a patient with tinnitus which was so distressing that he preferred total deafness, and was willing to run the danger of the operation rather than bear the noise longer. The operation, successfully performed, resulted in relief.

ENTOTIC NOISES.

Unlike the perversions of hearing, the examiner is able to hear the entotic noise. They are of two types: a clicking, snapping sound, and a blowing murmur, usually intensified with the cardiac pulsations. The clicking sounds may be due to some movement of the Eustachian tube or to spasmodic contraction of the tensor tympani. At times a patient is able to produce them at will, and thus becomes quite a curiosity. In other instances they are beyond the control of the will. The writer treated a patient throughout a year, during which time the snapping noise occurred with more or less regularity every one to five minutes. According to the statement of the patient, she was never free from the noise. By carefully observing the throat, it was possible to see a movement of the palate at the instant the snap occurred. It was therefore probably due to contraction of the muscles in relation with the Eustachian tube. A patient may have the noise only at irregular intervals. It is usually necessary to use the auscultation tube to hear these noises. Treatment seems to be of very little avail. Usually, however, the noise disappears of its own accord in time.

Vascular Murmurs.—The vascular murmurs may be due to enlarged arteries in the tympanum, to aneurysms in the intracranial arteries or of those in the neck. The following case, personally communicated by Dr. Don Campbell, of Detroit, is of interest in this connection: Male, aged twenty-two years, student, complained of inability to use his eyes. Had optic neuritis and bitemporal hemianopsia. Loud murmur at the ear which could be heard by the patient and also by the examiner. There was increasing deafness. It was believed that an aneurysm was present. The common carotid was tied with temporary improvement. Patient died three years later and autopsy revealed large cyst of the pituitary body pressing upon the carotid. It may be stated that the patient was under observation before the diseases of the pituitary body

were as well understood as they are today. Another patient recently seen, under the care of Dr. Alexander at the New York Eye and Ear Infirmary, presented a bruit which was audible through the auscultation tube, but much more plainly when the examiner's ear was placed in contact with the temporal region anterior to the auricle. X-ray examination shows enlargement of the sella turcica.

The prognosis of the vascular sounds varies with the condition causing them, and as much may be said of the treatment which is usually surgical, the nature of which must be determined by indications outside of the ear.

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